THE Journal of the Kansas Medical Society

JANUARY
1958

VOL LIX
NO 1

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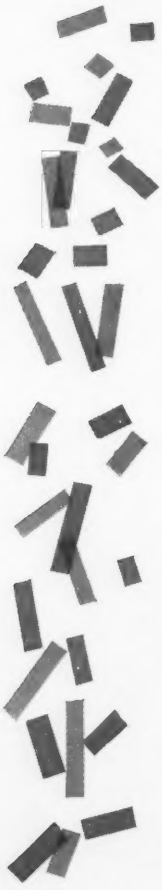
FOR PERSISTENT INFECTIONS

CHLOROMYCETIN®

COMBATS MOST CLINICALLY IMPORTANT PATHOGENS



Acquired resistance seldom imposes restrictions on antimicrobial therapy when CHLOROMYCETIN (chloramphenicol, Parke-Davis) is selected to combat gram-negative pathogens involving enteric and adjacent structures of the urinary tract. The acknowledged effectiveness with which CHLOROMYCETIN suppresses highly invasive staphylococci[1-9] extends to persistently pathogenic coliforms.[6,10-15] Experience with mixed groups of Proteus species, for example, "...shows chloramphenicol to be the drug of choice against these bacilli..."[15]

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

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(1) Petersdorf, R. G.; Bennett, I. L., Jr., & Rose, M. C.: *Bull. Johns Hopkins Hosp.* 100:1, 1957. (2) Yow, E. M.: *GP* 15:102, 1957. (3) Altemeier, W. A., in Welch, H., and Marti-Ibanez, F, ed.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 629. (4) Kempe, C. H.: *California Med.* 84:242, 1956. (5) Spink, W. W.: *Ann. New York Acad. Sc.* 65:175, 1956. (6) Rantz, L. A., & Rantz, H. H.: *Arch. Int. Med.* 97:694, 1956. (7) Wise, R. I.; Cranny, C., & Spink, W. W.: *Am. J. Med.* 20:176, 1956. (8) Smith, R. T.; Platou, E. S., & Good, R. A.: *Pediatrics* 17:549, 1956. (9) Royer, A.: Scientific Exhibit, 89th Ann. Conv. Canad. M. A., Quebec City, Quebec, June 11-15, 1956. (10) Bennett, I. L., Jr.: *West Virginia M. J.* 53:55, 1957. (11) Altemeier, W. A.: *Postgrad. Med.* 20:319, 1956. (12) Felix, N. S.: *Pediat. Clin. North America* 3:317, 1956. (13) Metzger, W. I., & Jenkins, C. J., Jr.: *Pediatrics* 18:929, 1956. (14) Woolington, S. S.; Adler, S. J., & Bower, A. G., in Welch, H., and Marti-Ibanez, F, ed.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 365. (15) Waisbren, B. A., & Strelitzer, C. L.: *Arch. Int. Med.* 99:744, 1957.

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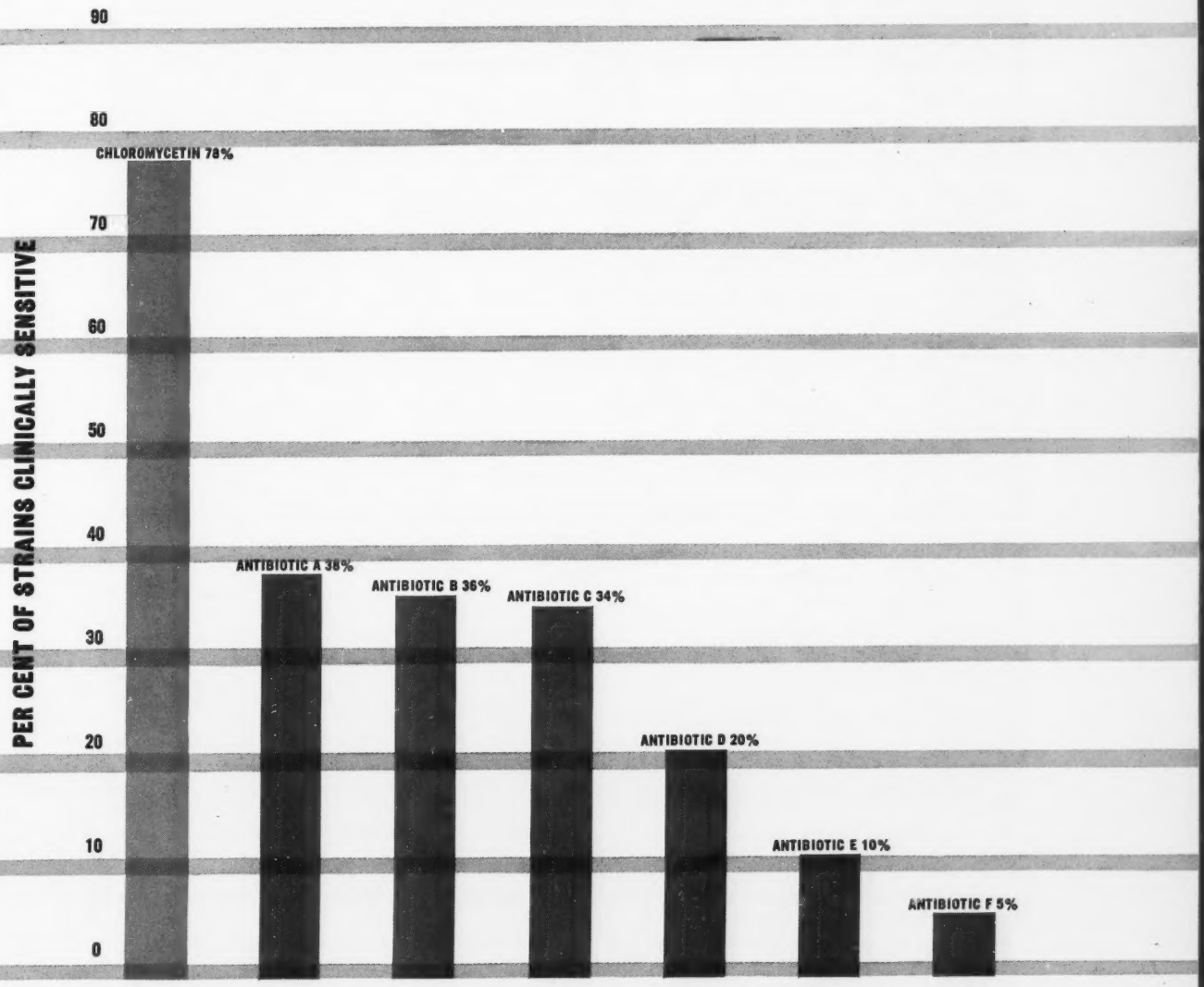

*This graph is adapted from Waisbren and Strelitzer.[15] It represents *in vitro* data obtained with clinical material isolated between the years 1951 and 1956. Inhibitory concentrations, ranging from 3 to 25 mcg. per ml., were selected on the basis of usual clinical sensitivity.

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Volume LIX JANUARY, 1958 Number 1

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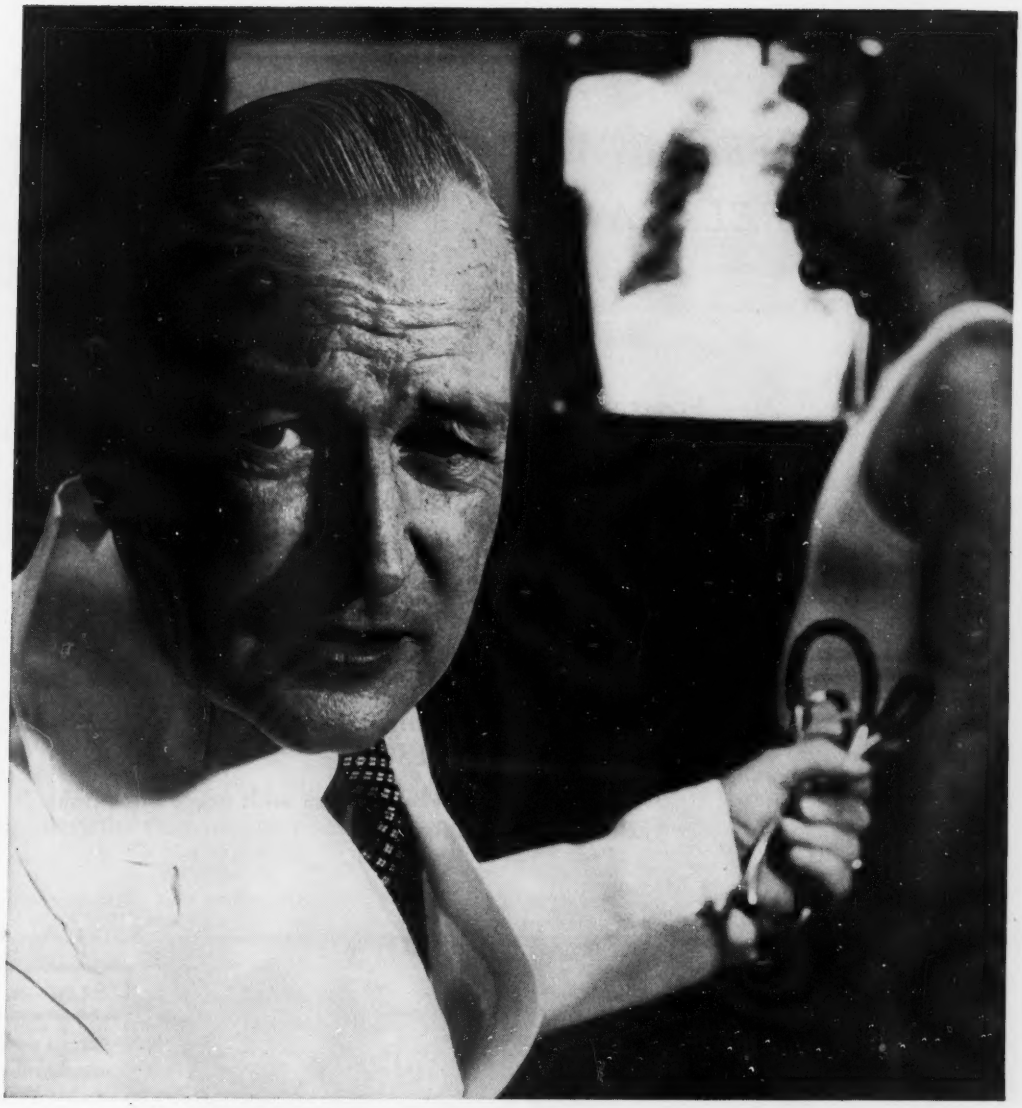

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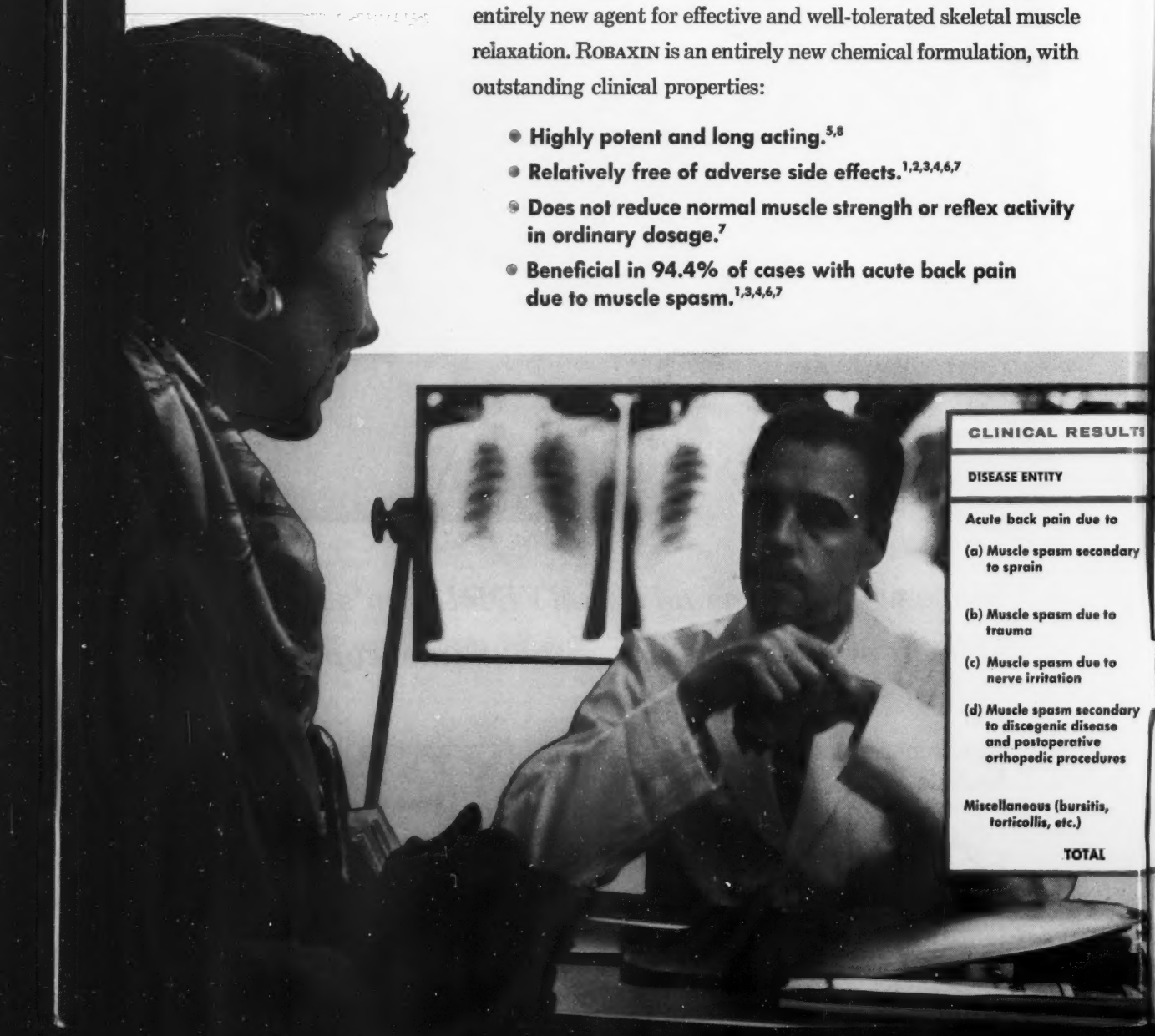

CLINICAL RESULTS	WI
DISEASE ENTITY	NO. CAS
Acute back pain due to	
(a) Muscle spasm secondary to sprain	18
(b) Muscle spasm due to trauma	13
(c) Muscle spasm due to nerve irritation	5
(d) Muscle spasm secondary to discogenic disease and postoperative orthopedic procedures	30
Miscellaneous (bursitis, torticollis, etc.)	6
TOTAL	72

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WITH ROBAXIN IN ACUTE BACK PAIN[1,3,4,6,7]

NO. OF CASES	DURATION OF TREATMENT	DOSE PER DAY (divided)	RESPONSE marked	mod.	slight	neg.	SIDE EFFECTS
18	2-42 days	3-6 Gm.	17	1	0	0	None, 16 Dizziness, 1 Slight nausea, 1
13	1-42 days	2-6 Gm.	8	1	3	1	None, 12 Nervousness, 1
5	4-240 days	2.25-6 Gm.	4	1	0	0	None, 5
30	2-28 days	1.5-9 Gm.	24	3	0	3	None, 25 Dizziness, 1 Lightheadedness, 2 Nausea, 2 *
6	3-60 days	4-8 Gm.	6	0	0	0	None, 6
72			59	6	3	4	*Relieved on reduction of dose

References: 1. Carpenter, E. B.: Publication pending. 2. Carter, C. H.: Personal communication. 3. Forsyth, H. F.: Publication pending. 4. Freund, J.: Personal communication. 5. Morgan, A. M., Truitt, E. B., Jr., and Little, J. M.: American Pharm. Assn. 46:374, 1957. 6. Nachman, H. M.: Personal communication. 7. O'Doherty, D.: Publication pending. 8. Truitt, E. B., Jr., and Little, J. M.: J. Pharm. & Exper. Therap. 119:161, 1957.

Indications – Acute back pain associated with: (a) muscle spasm secondary to sprain; (b) muscle spasm due to trauma; (c) muscle spasm due to nerve irritation; (d) muscle spasm secondary to discogenic disease and postoperative orthopedic procedures; and miscellaneous conditions, such as bursitis, fibrositis, torticollis, etc.

Dosage – Adults: Two tablets 4 times daily to 3 tablets every 4 hours. Total daily dosage: 4 to 9 Gm. in divided doses.

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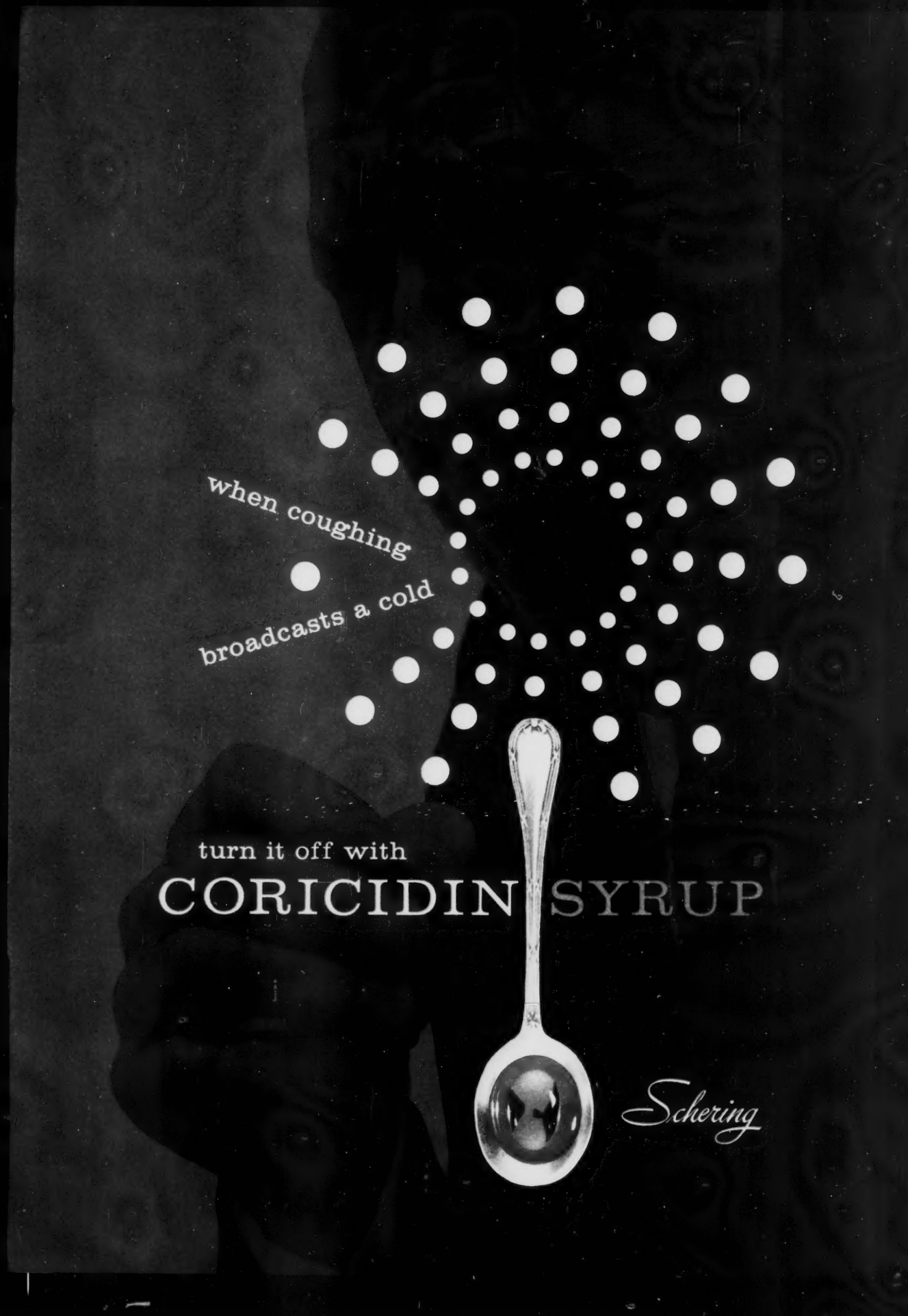
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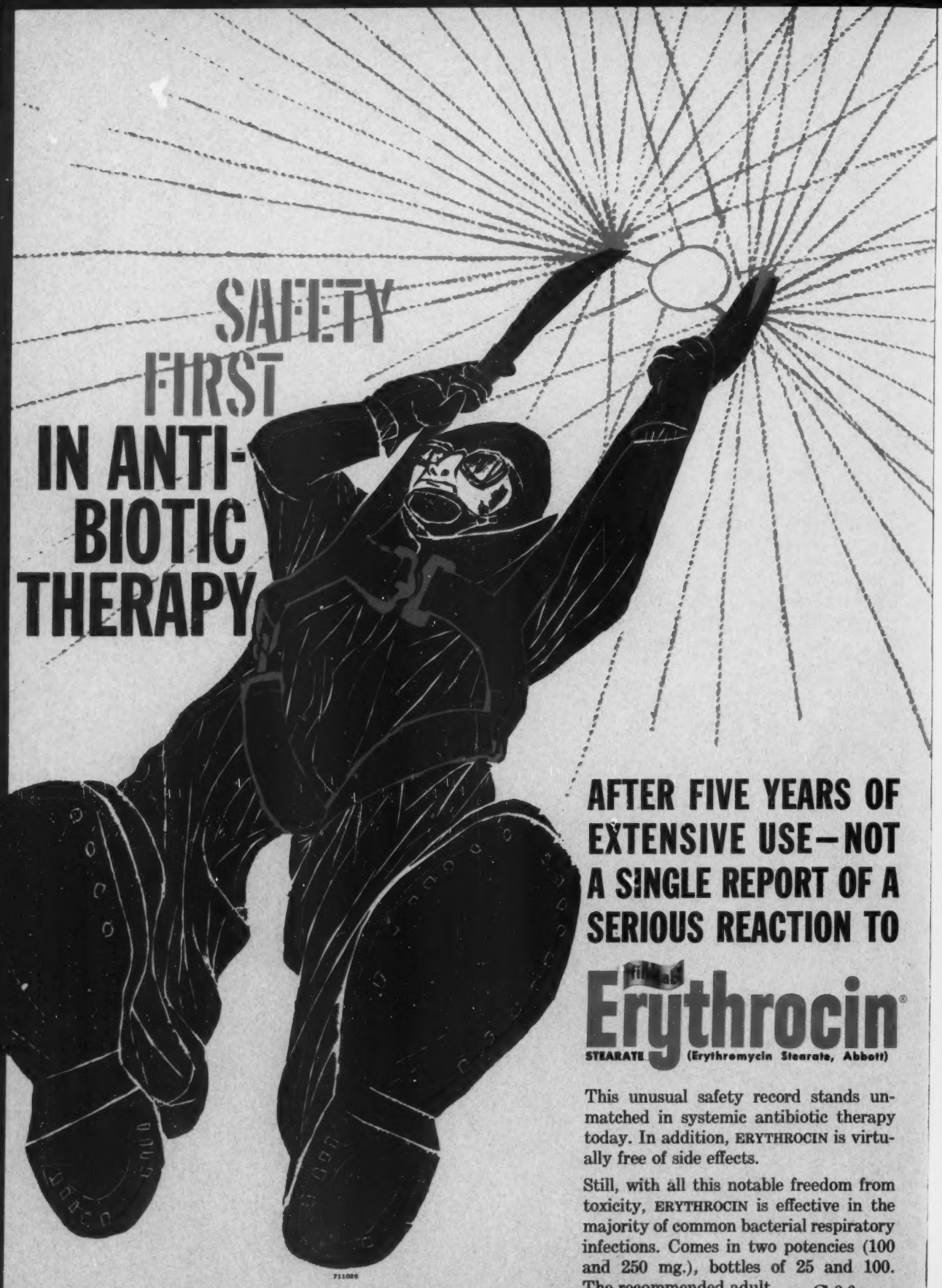

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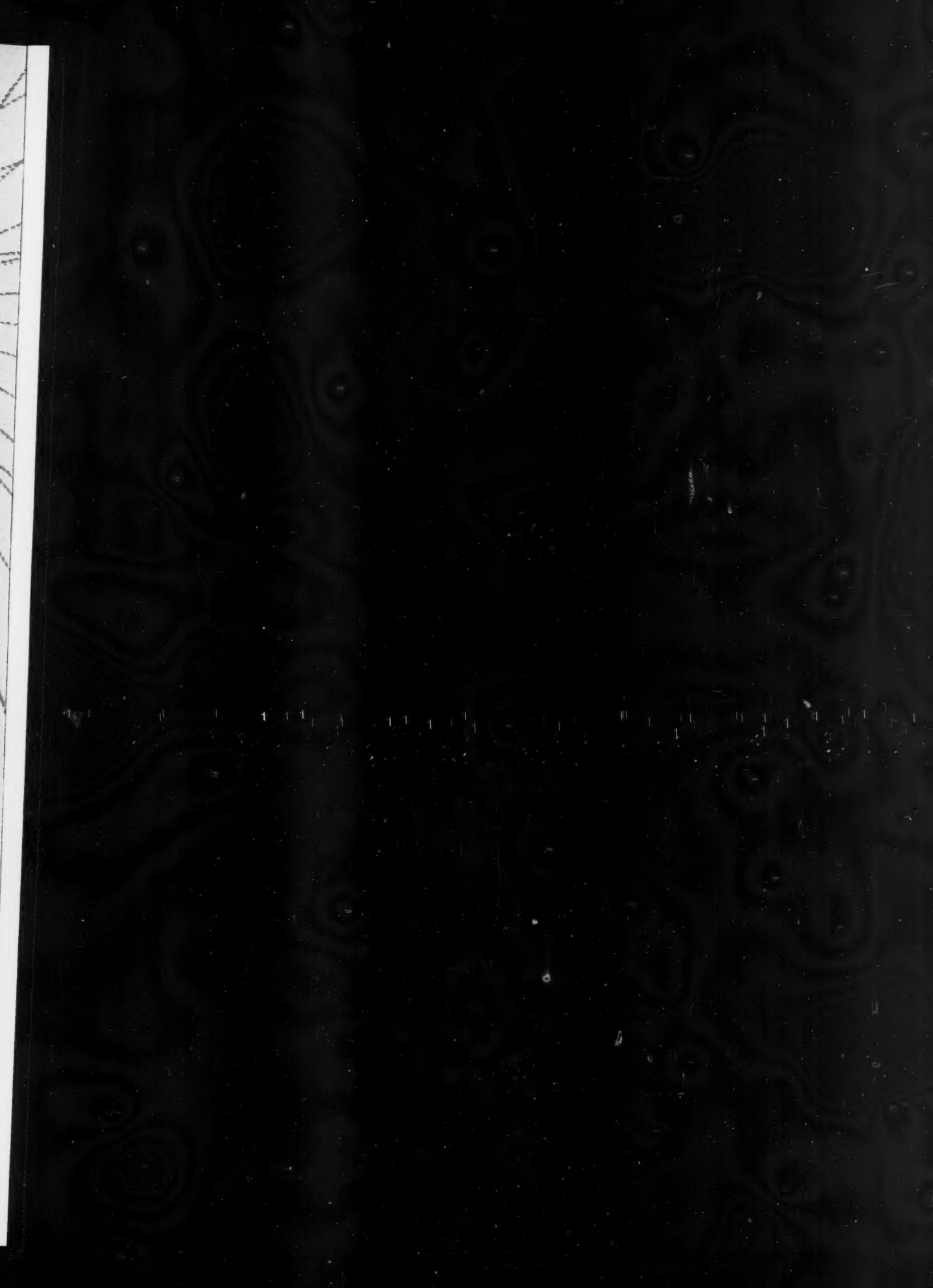

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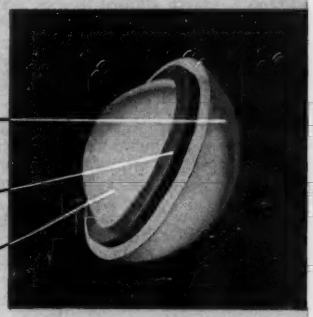

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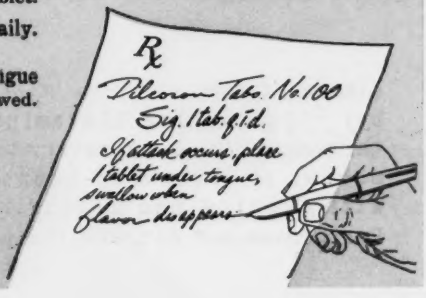
Therapeutic dose: **1 tablet held under the tongue until citrus flavor disappears, then swallowed.**



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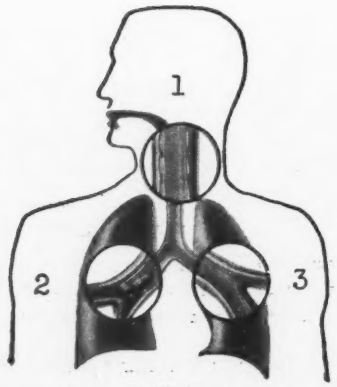

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Diagnosis	No. of Patients	Response				Side Effects
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Urticaria and angioneurotic edema	3	1	1	1		Dizzy (1)
Allergic dermatitis	2		1	1		Slight Drowsiness (2)
Bronchial asthma	1		1			
Pruritus	1		1			
Total	37	15	13	7	2	Drowsiness (5) Dizzy (1) 16.2%

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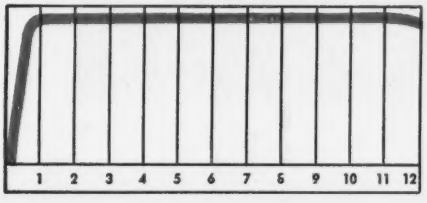

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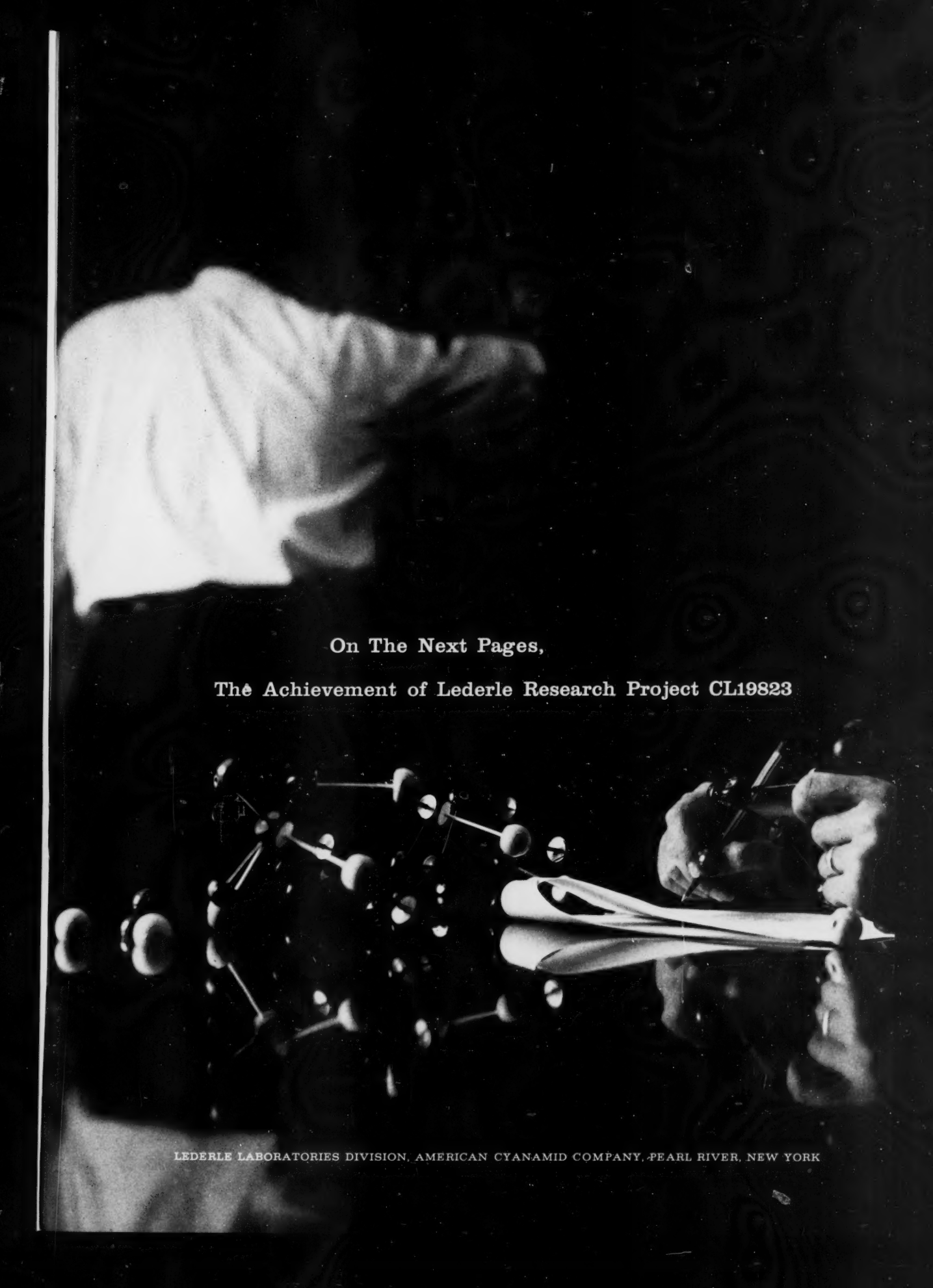

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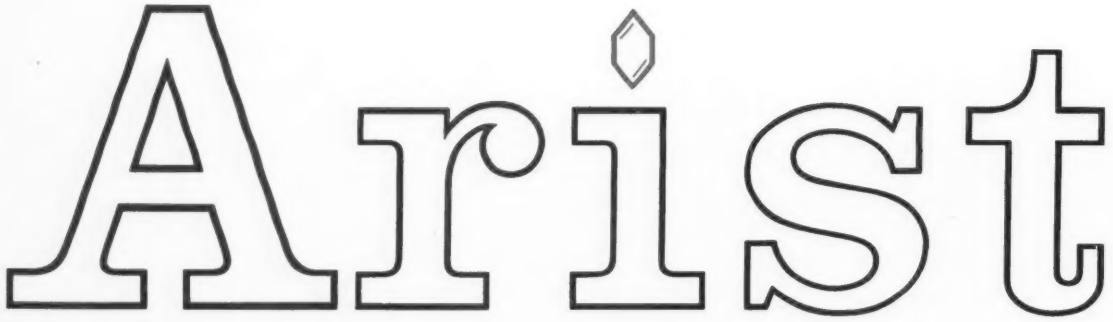

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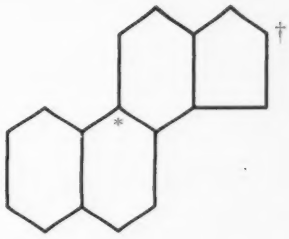

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- **a new high** in anti-inflammatory effects with lower dosage (averages 1/3 less than prednisone)
- **a new low** in the collateral hormonal effects associated with all previous corticosteroids
  - No sodium or water retention
  - No potassium loss
  - No interference with psychic equilibrium
  - Lower incidence of peptic ulcer and osteoporosis

Biological Effects of Aristocort

with particular emphasis on:

Kidney function

Animal studies on ARISTOCORT[1] have not demonstrated any interference with creatinine or urea clearance. Autopsy surveys of organs of animals on prolonged study of this medication have shown no renal damage.

Sodium and water

ARISTOCORT produced an increase of 230 per cent of water diuresis and 145 per cent sodium excretion when compared to control animals.[1] Metabolic balance studies in man revealed an average negative sodium balance of 0.8 Gm. per day throughout a 12-day period on a dosage of 30 mg. per day.[2] Additional balance studies showed actual sodium loss when ARISTOCORT was given in doses of 12 mg. daily.[3] Other investigators observed significant losses of sodium and water during balance studies and that those patients with edema from some older corticosteroids lost it when transferred to ARISTOCORT.[4,5] In two studies of various rheumatic disorders (194 cases) on prolonged treatment, sodium and water retention was not observed in a single case.[6,7]

Potassium and chlorides

There was no active excretion of potassium or chloride ions in animals given maintenance doses of ARISTOCORT 25 times that found to be clinically effective.[1] Potassium balance studies in humans[2,3] revealed that negative balance did not occur even with doses somewhat higher than those employed for prolonged therapy in rheumatoid arthritis. Hypokalemia, hyperkalemia or hypochloremia did not occur, when tested, in 194 patients with rheumatoid arthritis treated for up to ten and one-half months.[6,7]

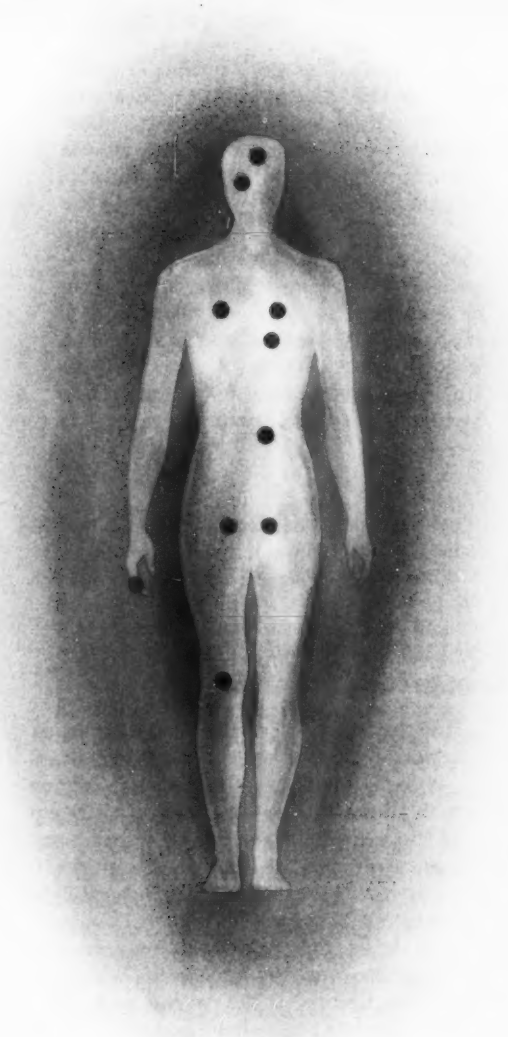
Calcium and phosphorus

Phosphate excretion in animals[1] was not changed from normal even with amounts 25 times greater (by body weight) than those known to be clinically effective. Human metabolic balance studies[3] demonstrated that no change in calcium excretion occurred on dosages usually employed clinically when the compound is administered for its anti-inflammatory effect. Even at a dosage level twice this, slight negative balance appeared only during a short period.

Protein and nitrogen balance

Positive nitrogen balance was maintained during a human metabolic study on maintenance dosage of 12 mg. per day.[3] At dosages two to three times normal levels, positive balance was maintained except for occasional short periods in metabolic studies of several weeks' duration.[2,3]

There was always a tendency for normalization of the A/G ratio and elevation of blood albumin when ARISTOCORT was used in treating the nephrotic syndrome.[8]



Liver glycogen deposition and inflammatory processes

An intimate correlation exists between the ability of a corticosteroid to cause deposition of glycogen in the liver and its capacity to ameliorate inflammatory processes.

In animal liver glycogen studies, relative potencies of ARISTOCORT over cortisone of up to 40 to 1 have been observed. Compared to ARISTOCORT, five to 12 times the amount of prednisone is required to produce varying but equal amounts of glycogen deposition in the liver.[1]

Most patients show normal fasting blood sugars on ARISTOCORT. Diabetic patients on ARISTOCORT may require increased insulin dosage, and occasional latent diabetics may develop the overt disease.

Anti-inflammatory potency of ARISTOCORT was determined by both the asbestos pellet[1] and cottonball[9] tests. It was found to be nine to 10 times more effective than hydrocortisone in this respect.

Gastric acidity and pepsin

The precise mode of ulcerogenesis during treatment with corticosteroids is not known. There is much experimental evidence for believing this may be related to the tendency of these agents to increase gastric pepsin and acidity—and this cannot be abolished by vagotomy, anticholinergic drugs or gastric antral resection.[10] Clinical studies[11] of patients on ARISTOCORT revealed that uropepsin excretion is not elevated. Further, their basal acidity and gastric response to histamine stimulation were within normal limits.

Central nervous system

The tendency of corticosteroids to produce euphoria, nervousness, mental instability, occasional convulsions and psychosis is well known.[12] The mechanism underlying these disturbances is not well understood.

ARISTOCORT, on the contrary, does not produce a false sense of well being, insomnia or tension except in rare instances. In the treatment of 824 patients, for up to one year, not a single case of psychosis has been produced. In general, it appears to maintain psychic equilibrium without producing cerebral stimulation or depression.

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The Promise of Aristocort in Reduction of Side Effects

It is axiomatic to affirm that the undesirable collateral hormone effects of corticosteroids increase in frequency and severity the higher the dosage and the longer used.

It has also become well recognized that the most serious of the major side effects from long-term corticosteroid treatment are peptic ulcers, osteoporosis with fracture, drug psychosis and euphoria, and sodium and water retention leading often to general tissue edema and hypertension.

It is significant that of the close to 400 patients on the lower dosage schedules found effective in bronchial asthma and dermatologic conditions, only 1 case of peptic ulceration has developed. No other of the above side effects have been observed even though ARISTOCORT was administered continuously to them for periods as long as one year.

The treatment of rheumatoid arthritis with steroids appears to result in the highest incidence of side effects. For this reason, the side effects associated with ARISTOCORT therapy in 292 patients with rheumatoid arthritis are below compared to the reported incidence of those from prednisone and prednisolone.

Peptic Ulcer

The most recent study available on the incidence of peptic ulceration in patients with rheumatoid arthritis on long-term prednisone therapy reported 12 ulcers in 49 cases (24 per cent).[1] Lowest incidence of 6.5 per cent has been recorded in a group of patients on this drug for six to nine months.[2] Four of six ulcers, in another series of 39 patients on prednisone,[3] appeared in less than three months of therapy.

The occurrence of peptic ulcer in 292 patients with rheumatoid arthritis treated continuously for up to one year with ARISTOCORT is approximately 1 per cent (2 of the 3 occurred in patients transferred from prednisone). In the remaining 532 cases recently analyzed, only one ulcer has been discovered in a patient who apparently had no ulcer when he was changed from another steroid.

Osteoporosis and Compression Fractures

The incidence of compressed fractures of vertebrae—and to a lesser extent in other bones—is high in patients on prolonged therapy with all previous corticosteroids.[4] One group of 49 patients[1] on long-term prednisone treatment experienced nine vertebral fractures (18 per cent); another series of 39 developed eight fractures (20 per cent),[3] four to 15 months after the beginning of steroid administration.

The occurrence of osteoporosis with compression fracture in 292 patients with rheumatoid arthritis treated continuously for up to one year with ARISTOCORT is 0.33 per cent (1 case[5]). Although these results are encouraging, determination of the true incidence of osteoporosis will have to await the passage of more time.

Euphoria and Psychosis

The euphoria so commonly produced by all previous corticosteroids has seemed a most desirable attribute to patients. In penalty, however, they have often later to pay for this by mental disturbances, varying from mild and transitory to severe depression and psychosis,[4] and toxic syndromes producing even convulsions and death.[6]

Since the onset of these complications is not directly related to duration of steroid administration,[7] the fact that not one case of psychosis occurred in 824 patients treated with ARISTOCORT, is most encouraging.

Sodium Retention—Hypertension—Potassium Depletion

When 17 patients were changed from prednisone to ARISTOCORT, 11 rapidly lost weight although only one had had visible edema.[8] Sodium and water retention, hypokalemia or hyperkalemia and steroid hypertension did not appear in 194 rheumatoid arthritis patients treated with ARISTOCORT.[5,9]

The interrelation between blood and body sodium, and steroid hypertension has long been generally appreciated.[10,11] Except in rare instances, or when unusually high doses are used (e.g., leukemia), the problem of edema and hypertension caused by sodium and water retention, has been eliminated with ARISTOCORT.

Minor Side Effects

Collateral hormonal effects of less serious consequence occurred with approximately the same frequency as with the older corticosteroids.[5] These include erythema, easy bruising, acne, hypertrichosis, hot flashes and vertigo. Several investigators have reported symptoms not previously described as occurring with corticosteroid therapy, e.g., headaches, lightheadedness, tiredness, sleepiness and occasional weakness.

Moon facies and buffalo humping have been seen in some patients on ARISTOCORT. However, ARISTOCORT therapy, in many instances, resulted in diminution of "Cushingoid" signs induced by prior therapy. Where this occurs, it may be related to reduced dosage on which patients can be maintained.

Reduction of dosage by one-third to one-half

In a double-blind study of comparative dosage in patients with rheumatoid arthritis,[12] 70 per cent of the cases were as well controlled on a dose of ARISTOCORT one-half that of prednisone. A general recommendation can be made that ARISTOCORT be used in doses two-thirds that of prednisone or prednisolone in the treatment of rheumatoid arthritis. There are individual variations, however, and each patient should be carefully titrated to produce the desired amount of disease suppression.

Comparative studies, of patients changed from prednisone, indicate reduced dosage of ARISTOCORT in bronchial asthma and allergic rhinitis (33 per cent),[8] and in inflammatory and allergic skin diseases (33-50 per cent).[13,14]

General Precautions and Contraindications

Administration of ARISTOCORT has resulted in a lower incidence of the major serious side effects, and in fewer of the troublesome minor side effects known to occur with all previously available corticosteroids. However, since it is a highly potent glucocorticoid, with profound metabolic effects, all traditional contraindications to corticosteroid therapy should be observed.

No precautions are necessary in regard to dietary restriction of sodium or supplementation with potassium.

Since ARISTOCORT has less of the traditional side effects, the appearance of sodium and water retention, potassium depletion, or steroid hypertension cannot be used as signs of overdosage. As a rule patients will lose some weight during the first few days of treatment as a result of urinary output, but then the weight levels off.

Patients do not develop the abnormally voracious appetite common to previous corticosteroid administration. In fact, some patients experienced anorexia, and it is advisable to inform patients of this and to recommend they maintain a normal intake of food, with emphasis on liberal protein intake.

While precipitation of diabetes, peptic ulcer, osteoporosis, and psychosis can be expected to appear rarely from ARISTOCORT, they must be searched for periodically in patients on long-term steroid therapy.

Traditional precautions should be observed in gradually discontinuing therapy, in meeting the increased stress of operation, injury and shock, and in the development of intercurrent infection.

There is one overriding principle to be observed in the treatment of any disease with ARISTOCORT. *The amount of the drug used should be carefully titrated to find the smallest possible dose which will suppress symptoms.*

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The Promise of Aristocort in Rheumatoid Arthritis

◊ ARISTOCORT *therapy has been intensely and extensively studied for periods up to one year on 292 patients with rheumatoid arthritis.*

Significant is the fact that most patients were *severe* arthritics, transferred to ARISTOCORT from other corticosteroids because satisfactory remission had not been attained, or because the seriousness of collateral hormonal effects had made discontinuance desirable.

Results of treatment

Freyberg and associates[1] treated 89 patients with rheumatoid arthritis (A. R. A. Class II or III and Stage II or III). Of these, 51 were on ARISTOCORT therapy from three to over 10 months. In all but a few patients, satisfactory suppression of rheumatoid activity was obtained with 10 mg. per day. Thirteen were controlled on 6 mg. or less a day, and for periods to 180 days. The investigators reported therapeutic effect in most cases to be A. R. A. Grade II (impressive) and that marked reduction in sedimentation rates occurred.

Another interesting observation in this study: Of the 89 patients treated, 12 had active ulcers, developed from prior steroid therapy. *In six patients, the ulcers healed while on doses of* ARISTOCORT *sufficient to control arthritic symptoms.*

Hartung[2] treated 67 cases of rheumatoid arthritis for up to 10 months. He found the optimum maintenance dose to be 11 mg. per day. Nineteen of these patients were treated for six to 10 months with an "excellent" therapeutic response.

Dosage and course of therapy

The initial dosage range recommended is 14 to 20 mg. per day—depending on the severity and acuteness of signs and symptoms. Dosage is divided into four parts and given with meals and at bedtime. Anti-rheumatic effect may be evident as early as eight hours, and full response often obtained within 24 hours. This dosage schedule should be continued for two or three days, or until all acute manifestations of the disease have subsided, whichever is later.

The maintenance level is arrived at by reduction of the total daily dosage in decrements of 2 mg. every three days. The range of maintenance therapy has been found to be from 2 mg. to 15 mg. per day—with only a very occasional patient requiring as much as 20 mg. per day. Patients requiring more than this should not be long continued on steroid therapy.

The aim of corticosteroid therapy in rheumatoid arthritis is to suppress the disease only to the stage which will enable the patient to carry out the required activities of normal living or to obtain reasonable comfort. The maintenance dose of ARISTOCORT to achieve this end is arrived at while making full use of all other established methods of controlling the disease.

ARISTOCORT is available in 2 mg. scored tablets (pink); 4 mg. scored tablets (white). Bottles of 30.

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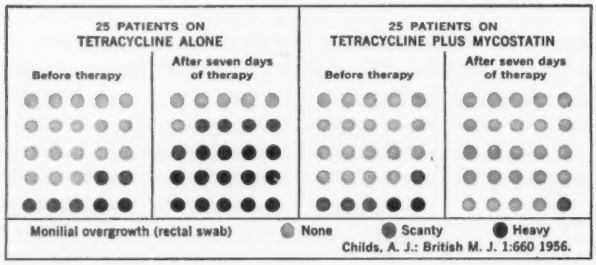

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1. Waldman, S., and Pelner, L.: Am. Pract. & Digest Treat. *8*:1075 (July) 1957.

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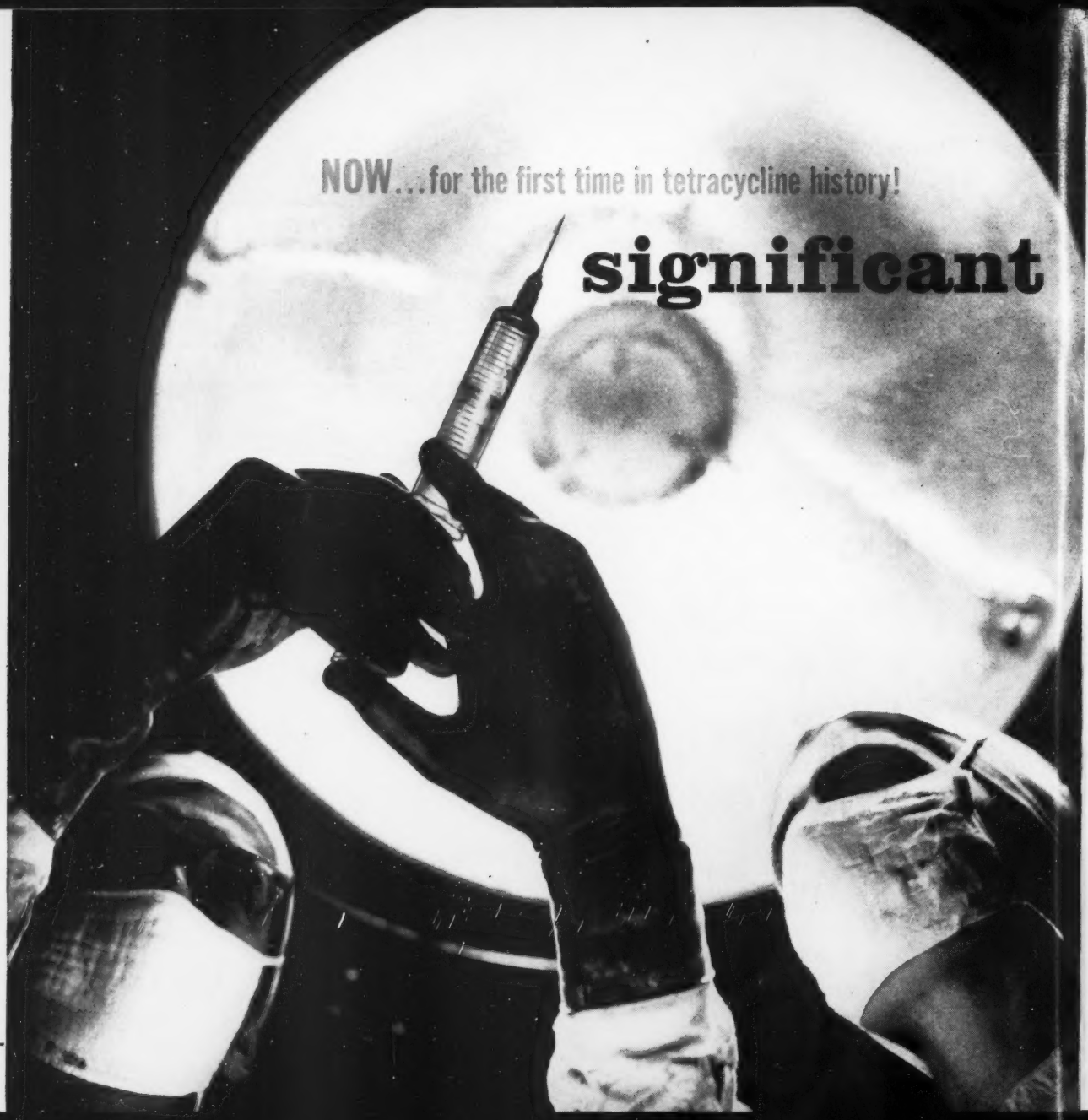

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Volume LIX **JANUARY, 1958** **No. 1**

Aortic Saddle Embolus

". . . one of the Few True Emergencies in Medicine . . ."

J. L. MORGAN, M.D., *and* **J. J. HOVORKA, M.D.,** *Emporia*

Saddle embolus of the aorta is a true medical emergency which is infrequent, but when it occurs it must be attacked surgically without delay. We are often alerted by the presence of an arrhythmia, particularly in mitral valvular disease, and more recently saddle embolus has been presaged by the pooling of blood during cardiotomy.

In the illustrative patient reported here with rheumatic mitral valve involvement and paroxysmal tachycardia, the complicated course was brightened by the fact that she was in the hospital when her embolus lodged at the aortic bifurcation, which permitted timely diagnosis and treatment.

The occurrence of a saddle embolus of the aorta with sudden blockage of the arteries to the legs is one of the few true emergencies in medicine. Too often we do not anticipate its occurrence, and all too often we fail to diagnose and act vigorously to save the legs or even the life of the affected patient.

Incidence

Actual incidence of this awesome complication is difficult to estimate, since, understandably, only successfully treated cases are dramatic enough to occupy space in medical journals. It has been reported that in Indianapolis, with 2,000 hospital beds, this problem is faced about three times yearly. In the 200 beds here in Emporia, in the past five years, there have been 11 peripheral emboli in addition to this solitary instance of a diagnosed aortic embolus. Five of these 11 were popliteal, three iliac, two femoral; one patient had multiple emboli. In different series, saddle emboli constitute between 5 and 15 per cent of all peripheral emboli. The recent increase in the number of operations on the heart is increasing the incidence of saddle emboli since they occur as postoperative complications.

Etiology

The embolus which lodges at the aortic bifurcation usually originates in the left side of the heart. Stasis of the blood flow, as in auricular fibrillation and in other arrhythmias, and the pooling of blood at cardiotomy are the most common background causes. Injury of the endocardium, as from a coronary occlusion, may present a sticky surface on which a clot forms, later to be dislodged and migrate to the iliac bifurcation. A general increase in the clotting ability of the blood has not been stressed as a prelude to the inception of saddle emboli.

The usual causes of aortic emboli are listed in Table I. Unusual causes are listed in Table II and are of interest because of their bizarre nature rather than their frequency.

Pathogenesis

The pathogenesis of this condition may be stated to be the formation of a clot or the existence of a

TABLE I

COMMON CAUSES OF AORTIC EMBOLI

1. Decrease in the velocity of cardiac blood flow
   a. Arrhythmias—especially auricular fibrillation in connection with rheumatic mitral stenosis with thrombus formation
   b. Cardiotomy with thrombus formation during cardiac immobility
   c. Congestive heart failure with thrombus formation, especially in the left auricle
   d. Rapid changes in heart rate due to digitalis therapy or its rapid withdrawal
2. Endocardial and valvular damage
   a. Disease of the aortic or mitral valves with thrombosis of the endocardium
   b. Myocardial infarction with thrombosis forming on the damaged myocardium and later dislodgment
   c. Dislodgment of mitral valve vegetations
   d. Sepsis of the umbilical cord in infants, with myocardial degeneration and thrombus formation

TABLE II

RARE CAUSES OF AORTIC EMBOLI

1. Decrease in the velocity of cardiac blood flow from arrhythmias such as auricular fibrillation *without* organic heart disease
2. From endocardial or vessel wall damage
   a. From calf vein phlebitis through a patent foramen ovale
   b. Dislodgment of a propagating thrombus in the pulmonary veins
   c. Migration of a pulmonary valve thrombus through a ventricular septal defect
   d. Thrombosis in an aortic aneurysm with emboli distally
   e. Thrombosis in a closing patent ductus arteriosus
   f. Migrating tumor fragments from a myocardial myxoma
   g. Syphilitic aortitis with dislodgment of plaques
   h. Migration of a bullet lodged in the heart
   i. Dislodgment of atheromatous aortic plaques
   j. Trichinosis of myocardium with thrombus dislodgment
   k. Tuberculosis of myocardium with thrombus dislodgment
3. From increased coagulation of the blood
   a. During closure of a patent ductus in a newborn baby with polycythemia and enhanced coagulability allegedly secondary to the polycythemia

foreign body proximal to the iliac bifurcation, which becomes dislodged from its moorings, travels in the aorta, and lodges at the Y of the bifurcation. The denial of the oxygen need of the legs leads to the classic changes of painful ischemia progressing to gangrene and loss of tissue, if the arterial block is not removed.

Diagnosis

The diagnosis is made by associating the predisposing cause such as a cardiac arrhythmia or commissurotomy with the so-called "five P's," namely Pain, Pallor, Paralysis, Pulselessness, and Paresthesias.

Pain has been blamed variously on the distention of the aorta, on arterial spasm distally, and on muscle ischemia. It is sudden and is the first symptom in at least half of the patients, though it may be absent for as long as several hours until ischemia sets in. Usually the pain is simultaneous in both legs. Ordinarily it is agonizing and radiates down the legs, though there have been a few instances when the onset was insidious with a sensation of pins and needles merging into the typical severe misery.

The pallor of aortic occlusion from a saddle embolus has been thought to differ from the "marbling" of the skin seen in aortic thrombosis.

Paralysis is common after the first few hours, but it may be overlooked because of the dominance of concern for the pain.

Pulselessness is the sine qua non for the diagnosis, and the absence of pulses from the iliac arteries down makes the diagnosis secure.

The paresthesias are characterized by numbness, coldness, and tingling in varying combinations.

The clinical picture of complete obstruction may change in a few hours if the embolus at the bifurcation breaks up and slips to a lower level, then presenting the picture of femoral or popliteal occlusion.

Differential Diagnosis

Conditions which might in occasional instances mimic saddle embolus of the aorta are listed in Table III.

Actually the sudden onset and rapid progression of

TABLE III

CONDITIONS OCCASIONALLY CONFUSED WITH SADDLE EMBOLUS

Thrombosis of the aorta, especially from trauma
Dissecting aneurysm
Arterial spasm accompanying thrombophlebitis
Acute intestinal obstruction (in youngsters)

leg pain, paralysis, paresthesias, and pallor in a patient with organic heart disease and arrhythmia, and the finding of pulselessness in the iliac arteries bilaterally makes the diagnosis of saddle embolus almost incontestable.

Treatment

1. *Surgical versus non-surgical management:* Evaluation of treatment depends on the definition of "cure" and the proof of cure. Criteria for "cure" have varied from the saving of life to complete restoration of circulation for at least three weeks postoperatively. Occasionally even the loss of leg tissue has not vetoed the classification as a "cure." Bauer[4] first successfully removed a saddle embolus in 1913, and in 1956 a survey reported 44 survivors following embolectomy, though of course embolectomy failures are not reported.

By 1954, in comparison, 21 patients were allegedly saved following conservative treatment, though only one well documented case, a patient later examined at autopsy, was reported.

Without surgical intervention the mortality has been listed as 75 to 95 per cent. As for aortic embolectomy, one series of 17 cases showed a mortality of 14, or 82 per cent.

From an exhaustive review of the literature it would appear that the great majority of observers advocate prompt surgical removal of the embolus once the diagnosis is made. There are a few who recommend non-surgical treatment either as a prelude to surgery or as a substitute for operation, particularly if there is a delay between onset and diagnosis (Table IV). *After* surgery, many of the measures listed in Table IV are widely used.

It is generally agreed that surgery should be performed as soon as the diagnosis is made, and to deliberately delay surgery while non-surgical measures are used would seem to be a breach of technic.

Age has been no absolute deterrent to surgery, one patient of 74 having survived operation.

TABLE IV
NON-SURGICAL MEASURES

1. Don't apply heat
2. Don't elevate the legs
3. Vasodilating drugs, e.g. papavarine
4. Anticoagulants
5. Sympathetic block with procaine
6. Sympathectomy
7. Buerger's exercises
8. Intermittent venous occlusion
9. Spinal anesthesia

One patient survived two aortic embolectomies for a period of five years.

It would seem that the conservative treatment of aortic embolism is surgical removal of the embolus as soon as the diagnosis is established.

2. *Timing:* It has been paraphrased that saddle emboli of the aorta should be treated with the same promptness as a perforated stomach ulcer, and this means preferably within the first six hours after onset. There is apparently no critical time, however, beyond which surgery should not be considered, since successful embolectomies have been reported at 25, 26, 32, and 33 hours after onset, and one recent case report detailed a successful embolectomy five weeks after the occurrence of the embolism.

3. *Anesthesia:* Spinal anesthesia has been the anesthesia of choice because it provides relaxation of abdominal muscles and a contracted state of the small bowel. One author does caution that spinal anesthesia may relax peripheral vessels and allow the embolus to migrate. Continuous spinal anesthesia with a Huber needle every four hours for 48 hours postoperatively was recommended in one report.

4. *The surgical approach:* A direct approach on the embolus may be made by an aortotomy, or the embolus may be approached indirectly through the iliac or femoral vessels. If an abdominal exposure is planned, the incision may be transperitoneal or retroperitoneal.

Actual exploration of the embolus makes more certain the complete removal of the clot. An extraperitoneal approach in obese patients occasionally has been thought to make for easier retraction of the intestines. The transperitoneal approach more readily permits a sympathectomy if this procedure is deemed advisable, though a retroperitoneal approach does supposedly allow accessibility to one sympathetic chain with a minimum of postoperative ileus. Actually the choice of transperitoneal or retroperitoneal approach rests with the surgeon.

Much has been written about the mechanism of occluding the aorta and iliac arteries during surgery. Many favor digital compression only, but other devices have been used such as rubber tubing, braided cotton tapes, Crile clamps, and Bethune lobectomy tourniquets. Certainly too vigorous constriction may fracture an arteriosclerotic plaque with subsequent distal embolism.

5. *Sympathetic block and sympathectomy:* Sympathetic block with procaine has been advised as a prelude to surgery, particularly if surgery is delayed, as a means of increasing peripheral circulation. On the other hand, there is some feeling that lumbar sympathetic block is contraindicated because the resulting vascular relaxation may allow an embolus to pass to

a more distal artery. There is admittedly a real danger in doing sympathetic block during anticoagulant therapy since hematomas have resulted from this therapeutic incompatibility.

Sympathectomy, either peri-arterial or of the chain itself, has been used at the time of surgery, and the desired accessibility of these nerve areas has influenced the type of exposure. Those advising against sympathectomy at the time of surgery point out that this procedure may result in a fall of blood pressure and a slower blood flow with an extension of the clot.

6. *Anticoagulants:* The majority of those with experience favor anticoagulants preoperatively, locally at the time of surgery, or postoperatively to prevent thrombosis distal to the embolus. As a result of occasionally annoying and dangerous bleeding, a few observers shy away from anticoagulants and use as a substitute reflex vasodilatation with heat and vasodilator drugs.

Those who advise heparin preoperatively do so only if sympathetic block and sympathectomy are not planned and advise neutralization of the heparin with protamine immediately preoperatively.

Those who use heparin at operation inject 50 to 100 milligrams into the aorta prior to closure of the incision.

Postoperatively heparin is usually continued intravenously every four hours until the prothrombin time is sufficiently prolonged by Dicumarol or similar drugs, and this usually takes one to three days. The duration of anticoagulant therapy with Dicumarol and similar drugs is an individual decision variously advised from three days to ten days, or even for years if the embolus follows coronary occlusion.

7. *Auriculectomy:* Amputation of the left auricular appendage has been advised after an embolectomy when it is felt the embolus arose in the auricle. Aortic embolism has occurred during adequate anticoagulant therapy with Dicumarol, and autopsy has subsequently disclosed the auricular appendage as the site of the embolus. Particularly has auriculectomy been considered indicated when the heart continues to fibrillate after embolectomy.

Mitral commissurotomy has been advised after an embolectomy in addition to auriculectomy when mitral stenosis exists, since auriculectomy has been felt to be inadequate alone if mitral stenosis exists. If auriculectomy is done, it should probably be more complete than that usually done at commissurotomy since in one patient an aortic embolus occurred two months after auriculectomy performed at the time of commissurotomy.

One recent article advises *emergency* mitral valvulotomy plus atrial appendectomy in patients with mitral stenosis prior to aortic embolectomy. This was successfully done in one patient with aortic embolism of six hours' duration, the time for the combined procedure being four hours and five minutes.

Complications

Enthusiasm for early embolectomy is considerably tempered by a long-time survey of the survivors of operation. In 1951 a review of 27 patients who had had successful embolectomies revealed that only two were alive after five years. The underlying cause of the embolism (see Table I) and the advanced age of many of the patients account for the poor long range prognosis.

In 9 of 16 cases, other emboli occurred before or after aortic occlusion. Widespread thrombosis may persist after embolectomy because of the accompanying spasm and intimal damage.

It is common for the distal pulses to take up to three weeks to return because of small distal emboli and slowness in formation of the collateral circulation. The patellar and Achilles tendon reflexes may take up to 12 months to return after embolectomy.

Case Report

Mrs. C. G., a 53-year-old white farmwife, was admitted to the hospital on October 21, 1955 and dismissed on November 30, 1955, a total stay of 41 days.

She was admitted at 2:00 p.m. An attack of rapid irregular heartbeat had developed suddenly at 8:00 a.m. the day of admission and persisted despite treatment.

Since 1940 she had experienced many bouts of paroxysmal tachycardia, and since 1948 she had been hospitalized six times for this difficulty, though her predominant rhythm was sinus in origin between attacks. She had been noted to have a grade ii systolic apical murmur in 1948, and this had persisted unchanged. She had her "inflammatory rheumatism" at age 14, lasting three months.

On physicial examination she appeared to be a pale, red-haired housewife lying comfortably in bed. Her blood pressure was 94/82. There was no goiter. Her apical heart tones were of a tic-tac quality, and the apex rate was 192 compared to a radial rate of 72. No murmurs were heard at this fast rate. Her peripheral leg pulses were patent.

A chest x-ray on admission showed no cardiac enlargement.

Her electrocardiogram on admission showed a supraventricular tachycardia with a rate of 180.

Five hours after admission the tachycardia converted to normal sinus rhythm following intravenous Vasoxyl therapy, and this rhythm prevailed for several days.

On November 1, 1955, nine days after admission,

at 7:15 a.m. when the patient was on her way to the bathroom to brush her teeth, she noticed sudden pain throughout both legs and the rapid onset of numbness and coldness. On examination at 7:30 a.m. both feet were found to be cold from the ankles down, and there was cyanosis of the nail beds. All leg pulses were absent. She could wiggle her toes only with difficulty. Three hours later the legs were cold from the mid-thigh down, and her pain had become agonizing. The diagnosis of an occlusion at the aortic bifurcation was made, and it was decided to proceed without delay with embolectomy which was begun at 11:20 a.m. (four hours after onset of symptoms) and was completed at 1:10 p.m. (duration one hour and 50 minutes).

The detailed procedure of the embolectomy was as follows:

Under spinal anesthesia (15 mgm. Pontocaine), the abdomen was opened through a lower midline transperitoneal approach. The small bowel was packed from the operative field. Direct examination revealed a well organized, firm, fixed embolus at the bifurcation of the aorta, extending upward 2.5 to 3 cm. and blunted Y-shaped tips extended about 1 cm. into the iliac vessels, forming a complete obstruction.

Figure 1.

Embolectomy was performed by incising the peritoneum 4 to 5 cm. directly over the embolus. The aorta above the embolus was mobilized by blunt dissection, sufficient to permit the passing of a soft penrose rubber tape (1 cm.) around it, to be used as a tourniquet. This was held in place by a right angle gallbladder clamp.

The aorta was incised longitudinally, about 2 cm., to permit the removal of the embolus. This was accomplished readily, and the smooth blunted ends indicated its complete removal. The tourniquet was momentarily released, flushing the proximal wound. A number 12 soft rubber catheter was passed downward into the iliac vessels and flushed with sodium citrate 2 per cent and Ringer's solution.

The aortic incision was closed with continuous 4-0 oiled silk, reinforced with silk mattress sutures. One hundred mgm. heparin in 10 cc. of saline was injected into the aorta, and the tourniquet was removed. A 1 x 3 in. Gelfoam pack was inserted over the suture line, and the peritoneum was closed with 3-0 chromic continuous sutures.

The abdomen was closed with chromic 2-0 continuous suture in the peritoneum, chromic 0 interrupted in the fascia, chromic 3-0 in the subcutaneous tissues, and dermal in the skin.

The embolus when removed appeared as shown in Figure 1 and measured 2 x 3.2 x 1.2 cm.

After operation the patient's legs were warm to touch and she could wiggle her toes with ease. The iliac arteries were palpable in both legs, but the more distal arteries were not pulsating. Her electrocardiogram postoperatively showed predominantly sinus rhythm with periods of sinus arrhythmia and a shifting pacemaker.

Five hours postoperatively there was bleeding from the lower edge of her dressing and this was estimated to be 150 cc. This was thought to be due to the heparin administered at the time of surgery. Dicumarol was started as soon as the patient was able to swallow postoperatively.

In the days following operation the patient developed several annoying arrhythmias varying from paroxysmal supraventricular tachycardia with delayed intraventricular conduction (November 3, 1955) to auricular flutter with varying degrees of block (November 12, 1955).

Despite what was felt to be adequate Dicumarol therapy, on the seventh postoperative day she began to cough and had pain in the left upper chest. A chest x-ray showed an area of linear density in the left upper lung field, and this was felt to represent a pulmonary infarct. Twenty-one days postoperatively she developed a tender indurated discolored area over the left greater saphenous vein, thought to be throm-

bophlebitis, again occurring during apparently adequate Dicumarol therapy.

Thirty days after operation, she was dismissed to her daughter's home, taking Dicumarol, with prothrombin times done twice weekly. Both feet were fairly warm. Both iliacs, popliteals, and the left posterior tibial artery were palpable. The right dorsalis pedis, right posterior tibial, and left dorsalis pedis arteries were not pulsating.

Her course at home was no less eventful than her stormy hospital stay. Six days after dismissal and 36 days after operation, she suddenly developed left arm and left leg weakness and a convergent strabismus. This was felt to herald a cerebral embolus originating in the heart, again occurring during adequate Dicumarol therapy. Her hemiplegia and strabismus cleared within three weeks except for a slight postural vertigo which still occasionally bothers.

She was continued on Dicumarol therapy for 80 days postoperatively.

Because of the possibility of mild hyperthyroidism as a contributory factor in her paroxysmal tachycardia, a radioactive iodine tracer study was done three months postoperatively, and this was reported as being within the normal range.

She was last seen April 24, 1957 as an office patient. She felt quite well except for mild postural vertigo. She was doing all of her own housework and was apparently as active as before her aortic embolus. She had had no bouts of tachycardia since surgery and was taking only maintenance doses of quinidine sulfate and digoxin. She still had a grade ii localized apical systolic murmur. The right foot felt slightly warmer than the left, though she was not conscious of any sensitivity to cold. The venous filling time in the right foot was 14 seconds and in the left foot 25 seconds. All leg pulses bilaterally were widely patent except the left dorsalis pedis artery.

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Anaphylactic Reaction

A Violent Reaction to Topical Penicillin

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Mrs. H., age 67, a white female, noticed a sore area in her mouth at 1:25 p.m. on September 3, 1955. She broke up a penicillin tablet and placed one-fourth of it on the raw area. Almost immediately her lip began to swell, and she complained to her husband of a numbness increasing in her legs. She felt cold and sweaty and began to faint.

He rushed her to our clinic, which took four to five minutes. As they traveled she complained of having increasing difficulty with her vision. On entering the waiting room she fainted. Her blood pressure was unobtainable, but her heart beat was regular, though faint to auscultation. Her skin was wet and clammy. She could not be roused by strong stimuli.

She was given Solu-Cortef, 100 mg. IV at 1:35; Adrenalin, 0.5 cc. subcutaneously at 1:40; Benadryl, 5 cc. intramuscularly, and Adrenalin, 0.3 cc. subcutaneously at 2:00; Adrenalin, 0.3 cc. subcutaneously at 2:15; Solu-Cortef, 100 mg. IV and Adrenalin, 0.3 cc. subcutaneously at 2:30. Her blood pressure was unobtainable during all this time, but the heart was beating regularly though faintly. Her breathing was not rapid but was very shallow. She did not respond to her name or to painful stimuli until 2:45. At that time her breathing improved and her blood pressure was 90/65. Since directions on Solu-Cortef suggest continuing with decreasing dosage, she was given 50 mg. intravenously at 4:30, 7:30, and 11:30 and 50 mg. intramuscularly at 11:30.

She was not hospitalized since she was kept at the clinic until 5:00 and was feeling quite well when seen at 7:30. She came in five days later and was given a B-12 1000 mcg. shot for moderate fatigue.

This case report describes a severe reaction due to penicillin, treatment administered, and data on the patients's recovery.

We saw the patient again in October, 1955, when she was having small areas of ulceration on her legs which were originally caused by insect bites. These responded to oral Vitamin C, 50 mg. four times a day, and A and D ointment locally.

The last occasion on which she was seen was in December, 1955, and that was for a small ulceration in her mouth, possibly herpes. This was handled with silver nitrate cauterization.

This case was of particular interest to me as I had been in practice for only 33 days at that time. We were pleased that the patient responded to treatment, even though the treatment had to be drastic. (Incidentally, I had not prescribed the penicillin for her.)

Chemotherapy in Tuberculosis

Report of Committee on Chemotherapy and Antibiotics
American College of Chest Physicians

General Considerations

At this writing there is no generally accepted optimum regimen in the chemotherapy of pulmonary tuberculosis. Streptomycin (SM), aminosalicylic (PAS) formerly para-aminosalicylic acid, USP XIV, and isoniazid (INH) are the three most commonly used drugs, but there is no unanimity of opinion as to which combination of these is most effective. However, it is emphasized that the best results are obtained when two or more drugs are combined and given continuously for a prolonged period of time. In general, it is probably unwise ever to treat a case of clinically active tuberculosis with one drug alone unless other drugs are contraindicated. Chemotherapy should be given for at least a year even in minimal cases and in advanced cases for a total of 18 to 24 months or at least until the stage of inactive disease is reached.

In all cases of tuberculosis, efforts should be made to culture the tubercle bacilli initially and to determine drug susceptibilities. This is essential in retreatment cases. Susceptibility studies are especially important if cultures remain positive, for changes in drug therapy may be based on changes in susceptibility.

Specific Drugs

The following drugs are useful in treating tuberculosis:

Isoniazid is a potent drug. It is effective at low concentrations, is readily absorbed, and penetrates all tissues of the body. It is easily administered and is relatively nontoxic with good patient acceptance. The most commonly accepted dosage of INH at the present time is 4 to 5 mg. per kg. of body weight daily, in two or three divided doses. It is estimated that some individuals will have inadequate serum levels of INH as measured by bio-assay on this dosage level. Evidence is at hand that about 85 per cent of patients with new tuberculosis will do well on standard doses of INH (300 mg. per day) in combination with other effective drugs. In the other 15 per cent, particularly in patients with more advanced disease with large or multiple cavities, it is probably advisable to individualize the dosage of the drug with consideration given to higher dosage. Toxic effects of this drug, particularly peripheral neuritis, are commoner at the higher levels and pyridoxine (100 mg. per day) must be administered concurrently whenever the higher dosages are to be used. Hypersensitivity reaction may occur in the use of this drug as with streptomycin or PAS.

There are two major facts to be kept in mind in the use of INH: (1) As with most of the other effective drugs the tubercle bacilli readily becomes resistant to this drug when it is administered alone; (2) Isoniazid is degraded in human subjects into several derivatives such as acetyl-isoniazid which are biologically inactive; such inactivation varies significantly from individual to individual. Serum levels of this drug determined by the standard chemical methods will not reveal the inactivation, but it will be evident if bio-assay methods are used.

This report, released by Murray Kornfeld, executive director of the American College of Chest Physicians, is intended as a progress report or statement on currently accepted principles and practice, not as a detailed treatise for chemotherapy. It may be used as a guide by the physician treating tuberculosis.

Streptomycin and Dihydrostreptomycin continue to be among the most effective antituberculosis agents at our disposal. Each has the same therapeutic value and the dosage is the same for both. They are generally administered in a dosage of at least 1 gm. twice weekly by intramuscular injection. In this dosage streptomycin rarely causes vestibular damage and dihydrostreptomycin rarely results in deafness. In an effort to avoid these rather remote possibilities some physicians prefer a combination of streptomycin 0.5 gm. and dihydrostreptomycin 0.5 gm. In studies reported by the British Medical Research Council it was evident that, when administered in combination with daily INH, streptomycin was more effective in preventing the emergence of INH resistant organisms when given in daily dose of 1 gm. as compared with dose of 1 gm. twice weekly. Preliminary reports are appearing indicating that in some patients, particularly those with advanced disease, intermittent streptomycin may be less effective than daily administration of 1 gm. of this drug. It may be advisable to give streptomycin in doses of 1 gm. daily for at least 30

days to a patient severely ill on admission before reverting to intermittent therapy. Hypersensitivity to streptomycin occurs occasionally and is manifested by fever, rash and sometimes exfoliative dermatitis. In patients with less severe reactions desensitization may be accomplished by starting with a very small dose and gradually increasing; with more severe reactions desensitization may be hazardous and probably should not be attempted. Occasionally, a patient hypersensitive to streptomycin may be able to tolerate dihydrostreptomycin and vice versa.

Aminosalicylic Acid remains an important agent in the antimicrobial therapy of tuberculosis due to its ability to prevent or postpone resistance to streptomycin and INH; and to its ability to enhance the serum levels of active INH. Many forms of this drug are on the market from the acid product to sodium, potassium and calcium salts of the acid, a buffered product, and other forms. The dosage for all of these must be adjusted to the dose of the acid. In other words, 15 gm. of sodium PAS is the equivalent to 12 gm. of acid PAS. Many patients will have less gastrointestinal intolerance on some one of these products than on others. There is some difference in blood levels produced with these drugs. Sodium and potassium PAS being rapidly absorbed have rapid peaking and falling off of blood levels, while with other forms a more prolonged peak may be attained. The clinical significance of this is undetermined at the present time.

PAS preparations of all types if stored too long or exposed to undue heat, light or moisture, deteriorate and discolor, resulting in increased intolerance or actual toxicity. PAS should be prepared fresh if given in solution. Under best conditions, side reactions of anorexia, nausea and diarrhea are not uncommon with all forms of PAS, but are not necessarily indications for discontinuing the drug. Occasional patients develop more severe reactions with fever, rash and rarely with severe systemic reactions simulating infectious mononucleosis.

PAS alone is relatively not very effective as a treatment for tuberculosis and should always be used in combined therapy. It has been shown recently that PAS, when administered concurrently with INH, will enhance the level of free INH in the serum of patients who rapidly inactivate INH. In Europe intravenous PAS is being used extensively and claims have been made for its value by this route.

The standard dose of PAS in this country is 12 gm. daily in three divided doses, although some studies have indicated that smaller doses of the active substance may well be useful, particularly if full dosage is not tolerated.

Viomycin has a useful though rather limited place in the treatment of the patient whose organisms are resistant to isoniazid and streptomycin and for whom an umbrella is desirable for resectional surgery. The usual dosage is 2 gm. (IM) twice weekly for two or three weeks before surgery and eight to ten weeks or more postoperatively. When feasible it should be combined with another drug to which the organisms are sensitive. Renal toxicity precludes the daily use of this drug, but is less evident when used twice weekly.

Pyrazinamide (PZA) is now undergoing clinical investigation by the Veterans Administration-Armed Forces group, the USPHS group, and others, particularly in combinations with isoniazid. It has been found to be effective in combination with INH when administered to patients who have never received either drug before. There is some evidence that this drug may be effective for short periods of 30 to 60 days when used alone, particularly to cover resectional surgery in patients resistant to the other major drugs. In most studies reported, there has been a significant factor of toxic effect on the liver; approximately 10 per cent of the patients receiving pyrazinamide have shown abnormal results in liver function studies and about 3 per cent have shown frank jaundice. When this drug is administered liver function studies should be done periodically to estimate any liver toxicity. Most of the toxic conditions resulting from the use of this drug, however, revert to normal when the drug is withdrawn. PZA should be discontinued promptly if significant disturbance in liver function is noted and invariably if jaundice appears. At the present time, due to severe toxicity of the drug, it should be administered only to patients in the hospital. This drug is ordinarily administered in dosage of from 30 to 40 mg. per kg., orally administering no more than 3 gm. daily. Hyperuricemia has been reported in conjunction with the use of PZA.

Cycloserine is a relatively new antibiotic under investigation for use in the treatment of tuberculosis. Preliminary studies have shown that this drug used alone is not as effective in the treatment of tuberculosis as are the various combined drug regimens now in use. At present, studies are in progress to determine the effectiveness of this drug when used in combinations with INH. Reports of toxicity, particularly to the nervous system, have continued such as tremors, drowsiness, convulsions and psychoses. Most investigators originally used this drug in dosage of 1 gm. daily, orally, in divided doses. Newer studies indicate a maintenance of therapeutic effectiveness and nearly complete absence of toxicity when administered in doses of 0.25 gm. twice daily in combination with isoniazid.

Recommended Regimens: Though there is no generally accepted optimum chemotherapy regimen for

pulmonary tuberculosis at the present time recent reports of the Veterans Administration-Armed Forces group and of U. S. Public Health Service sponsored studies indicate that the following regimens give approximately the same clinical results in most cases of tuberculosis: (1) Isoniazid, 300 mg. daily plus PAS 12 gm. daily; (2) Isoniazid 300 mg. daily plus SM 1 gm. twice weekly, and (3) Isoniazid 300 mg. daily plus SM 1 gm. twice weekly plus PAS 12 gm. daily. The Veterans Administration and U. S. Public Health Service studies indicate that the regimen of streptomycin 1 gm. twice weekly and PAS 12 gm. daily is not quite the equal of the other three regimens, and that in far advanced disease with large cavities INH-PAS is superior to intermittent SM-INH.

As has been pointed out above, there is increasing evidence that the drug regimens must be individualized in certain patients, particularly in those with more advanced disease, with larger doses of INH and daily SM being administered as indicated.

Acute Miliary Tuberculosis

Isoniazid has proved to be very effective in the treatment of miliary tuberculosis with survival rates of 90 per cent and higher being reported. Any standard INH containing combined regimen should be adequate in treating this condition, but due to the serious nature of miliary tuberculosis many still advocate the use of triple drug therapy with higher dosages of isoniazid such as 10 mg. per kg. per day being used. The drug therapy should be continued for at least 18 months.

Tuberculous Meningitis

Reports during the past several years indicate that survival rates of 80 per cent to 90 per cent or higher are possible in tuberculous meningitis when INH, SM and PAS are administered for a minimum of 24 months. The Committee suggests a dosage schedule similar to that for miliary tuberculosis. Intrathecal medication is not recommended. It is of the utmost importance to start the treatment immediately if the history, physical examination or spinal fluid findings strongly suggest a diagnosis of tuberculous meningitis. If the patient's condition does not permit oral medication, the INH and PAS may be given parenterally, initially.

Genitourinary Tuberculosis

Genitourinary tuberculosis responds very well to combined drug therapy including INH, SM and PAS in dosage as recommended for pulmonary tuberculosis. The drug should be administered for 18 to 24 months. Recent reports from the Veterans Administration-Armed Forces study indicate that long-term therapy with INH, SM and PAS is very often definitive in such cases and the need for surgical intervention is becoming surprisingly less frequent.

Tuberculosis in Childhood

The Committee recommends that all children with active primary tuberculosis should receive antimicrobial therapy. The complications such as miliary and meningeal tuberculosis which sometimes occur in primary disease have sharply declined since the advent and use of INH. Consideration should be given to the treatment of recent tuberculous converters, particularly in children under four years of age. In children with active tuberculosis, the physician should always be on the alert for the development of miliary or meningeal tuberculosis. The approximate dosages of the antituberculosis drugs for children are as follows: SM 30 to 40 mg./kg. twice weekly, INH 10 to 16 mg./kg./day and PAS 200 mg./kg./day. Children tolerate higher dosages of INH well and administration of pyridoxin is usually not needed to prevent toxicity.

Other Forms of Tuberculosis

When the disease involves such organs and tissues as the larynx, mouth, lymph nodes, trachea, bronchi, GI tract and bone it is best treated by long term combined chemotherapy using one of the regimens recommended for pulmonary tuberculosis.

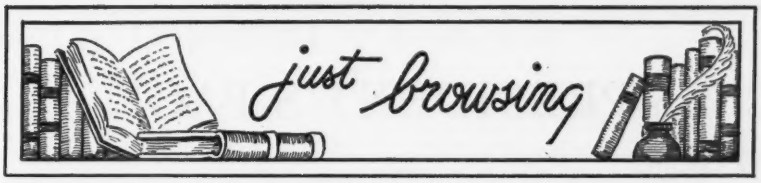
Tuberculous Pleurisy with Effusion

This condition should be treated as a case of acute pulmonary tuberculosis with long term continuous combined chemotherapy for a year or more. This recommendation also applies to the so-called idiopathic pleurisy with effusion patients with a positive Mantoux even though careful studies fail to reveal presence of tubercle bacilli in the pleural fluid. Experience has shown that in such cases the etiology is usually tuberculous and should be treated as such in order to avoid reactivation later.

Steroid Therapy in Tuberculosis

The exact role of cortisone and related compounds in the management of infectious diseases is undefined. However, the greatest difference of opinion regarding the place of steroids exists in the field of tuberculosis. Some have felt that this form of therapy is always contraindicated while others have recommended its use under certain specific circumstances. Some of the tissue damage and clinical manifestations in tuberculosis are due to an exaggerated interaction between sensitized tissue and tuberculoprotein. Corticosteroids may suppress this overactive defense mech-

(Continued on Page 31)



Did you ever wonder how "adopted parents" feel toward their children, or whether they feel any different than "natural parents"? The author of a little poem entitled "Our Baby," again an unknown individual, has beautifully expressed the attitude of parents who have taken into their homes children in less fortunate circumstances.

Some day I'll have to tell you, dear,
by birth you're not my own . . .
Or else some meddler will impart the
knowledge which is known,
That we adopted you, to start a home
for us alone.

I'll try to make it plain to you and
pray you've understood,
Our friends all had a child or two,
their lives seemed full and good,
So . . . there was nothing left to do,
but find you if we could.

You see, God did not hear my prayer
and let me bear a child,
My heart was sick with longing,
dear, until the day you smiled,
And snuggled close within my arms
—then you became my child.

It hurts my heart when people say,
"She's really not your own" . . .
They cannot ever guess the love for
you that fills our home,
You were not born to me, that's true
. . . but birth's so small a part,
Of Motherhood . . . and daily tasks . . .
which fill a mother's heart.

It's we who've known the agony of
terror stricken night . . .
And prayed, "Oh, cool her fever, God,
and please make her all right."
It's we alone who knew the joy of
teaching you to walk . . .
We've picked you up and kissed you lots
and thrilled when first you talked.

No natural parents have a voice in
choosing what they get . . .
A boy . . . a girl, there's no choice
of blonde or dark brunette.
So, I hope you'll be happy, dear, and
have no vain regrets . . . for . . .
From all the babies in this world I
sought you for my own . . .
Yes, you're mother's "Chosen Child"
. . . my child . . . and mine alone.

When asked to tell a group of schoolgirls in the slums of one of our large cities how to be happy, Alice Freeman Palmer, one of the earlier presidents of Wellesley College, told them: "I will give you my three rules for being happy, but mind, you must all promise to keep them for a week, and not skip a single day, for they won't work if you skip one single day. The first is that you will commit something to memory every day, something good. It needn't be much—three or four words will do—just a pretty little poem or a Bible verse. The second rule is, look for something pretty every day and don't skip a day or it won't work—a leaf, a flower, a cloud—you can all find something. My third rule is—now mind, don't skip a day—do something for somebody every day."

* * *

We all have our ideas of what friendship is, but some unknown individual summed it all up in this paragraph. "Friendship is a plant which requires careful cultivation. If it is well cared for it may grow to be a real century plant, blooming and bearing fruit through generations. To attain perfection it must be watered with the tears of sympathy, invigorated with the sunshine of happiness shared, protected from the parasites of envy and selfishness, and the weeds of distrust and discord must be eliminated. It should be sheltered from the frost of indifference and criticism, and it must be hedged about with a fence of trust and devotion."

* * *

God hath not promised
Skies always blue
Flower strewn pathways
All our lives through,
God hath not promised
Sun without rain
Joy without sorrow
Peace without pain.

But God hath promised
Strength for the day,
Rest from labor,
Light for the way,
Grace for the trials,
Help from above,
Unfading sympathy,
Undying Love.

* * *

May the New Year 1958 bring to each of you a full measure of the really worthwhile things of life.—*O.R.C.*

PRESIDENT'S PAGE

DEAR DOCTOR:

Your president, I fear, has disturbed our medical colleagues of the "effete East."

It was in Philadelphia at the recent A.M.A. meeting during discussions in the Reference Committee on the pending legal action of the United Mine Workers against the Colorado Medical Society. Organized medicine in Colorado wants to bar its members from cooperating in the U.M.W. closed panels for medical care of the miners and their families. Apparently such boycotting of doctors is acceptable to the profession.

Not acceptable, however, was the shocking suggestion of the discussant from Kansas, who opined that such situations should be resolved at the very start of negotiations by requiring the unions to deal with medical society representatives in preparing their medical plans. That they should not be permitted to dicker with single doctors, who, for one reason or another, can be maneuvered into a form of medical practice which is counter to accepted principles of proper patient-doctor relationships, and counter to approved modes of furnishing medical service.

This, said our confreres from eastern states, would be beneath the dignity of the medical profession, would be stooping to trade union levels, would be nothing more or less than collective bargaining. So what? Is there valid objection to collective action to preserve what we sincerely regard as inviolable principles; to preserve the free, unshackled accomplishment of the mission to which we are dedicated as physicians?

Our friend, the governor, has likened us to the teamsters' union. He seems to exhibit a measure of respectful regard for a "tight" organization. As he should. And so should the unions or any other organization dealing with medicine.

It is submitted that there should be in each state medical society, as well as in our national association, a body of men, representative, competent, carefully selected men, to act as a committee; or possibly they should be referred to as a commission. Their function: to serve as the negotiating representatives of organized medicine through whom, and only through whom, could be negotiated the preparation of any and all plans for group medical care.

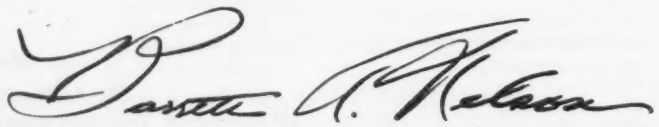
This, in all probability, the unions would not approve. Unquestionably they realize that on the "divide and conquer" principle they would have little difficulty finding an ample number of individual doctors willing to compromise the principles we have established and supported. But logically, of course, any stand opposing collective action is completely untenable to an organization whose entire purpose in being is just that.

Certain legal points must be considered. (The A.M.A. once was haled into court for "restraint of trade.") These points should not be insurmountable and should be susceptible to solution by the astute attorneys in the A.M.A. Legal Department.

It is only a matter of time until this situation will arise in Kansas. The more one thinks of the above proposal, the more practical and logical it seems to become.

Let's give it serious consideration. Maybe something will come of it.

Fraternally yours,



President

EDITORIAL COMMENT

Best Jokes of 1957

The past year probably was much like any other with its share of success and of failure. There was a little humor also if you will order it slightly rare.

There was the Russian trick of floating a dog in a satellite, which became funny when the United States blew trumpets and announced it was our turn. We should have had Dizzy Dean pitching that day.

There was a repeat performance of the melodrama entitled "Who's Got the Vaccine?" which was played a few years ago by a slightly different cast. Uncle Sam retained the lead of course because as its angel he could control the production.

This too had its lighter side. As long as "tickets" were scarce, everybody wanted them. Outrageous scalping activities even got into the paper. Then when news got around that the shortage was over, you couldn't give them away.

Kansas passed a Basic Science Act and got a composite Healing Arts Board and examined osteopaths in medicine and surgery. If there is any real humor in this event, it has been shown so far only by a couple of newspaper editors and by a very few other persons, such as the legislator who said if he had known the Basic Science Act would prevent anyone from practicing in this state he would have voted against it.

This little comedy gets better in a subsequent act where 200 big town doctors suddenly become available for 200 western Kansas communities—except for their reluctance to face the Basic Science Board in this state.

During 1957 at least, these doctors do not appear in the play. So far the speaking parts are confined to a monologue on the part of an editor. He was abetted by some political pantomiming but by and large the whole business was not very well staged.

Not that these problems aren't serious. They are. The standards of health care, the problems of physician distribution are great, and they are important. Their solution requires the cooperative effort of many persons in many fields of endeavor.

They are being solved in many places of Kansas where the people and the medical profession, and yes, the newspaper editor have all combined their efforts. The Kansas Medical Society takes great pride in its contributions toward supplying Kansas with the finest health care available anywhere and can produce evidence to support the outstanding position Kansas enjoys in this regard.

The comedy arises, if you like that type of humor, where one little man decides he will solve the problem all by himself. And last year gave us a couple of examples like that.

Group Life Insurance

The American United Life Insurance Company of Indianapolis will open enrollment for group life insurance for all members of the Kansas Medical Society under the age of 70 years on February 1 and will continue an open enrollment for 60 days thereafter.

Insurance for every physician who signed an application (all under 70 years of age) will become effective on February 1 without the necessity of establishing physical insurability. After the 60-day period, enrollment will be limited to those who can physically qualify, except for new members of the Society who will have a six-months enrollment eligibility.

The Kansas Medical Society is pleased to announce that the plan for making group life insurance available to its members has finally succeeded. This has been studied for years by the Committee on Medical Economics and has been endorsed by the House of Delegates. There has just now been sufficient interest to place the plan into effect.

In the briefest analysis the Kansas group life program is term insurance from purchase date to age 70. The premium is $100 a year. The face value is highest at earlier ages and decreases annually. It may be converted to other forms of insurance without proof of insurability.

The Kansas Medical Society believes this to be a benefit to its members and of especial value to young physicians. It is strongly urged that each doctor examine this program. More information may be obtained from the Kansas Medical Society, 315 West Fourth Street, Topeka, Kansas.

H.R. 9467

During the first session of the 85th Congress Mr. Forand, a Democrat of Rhode Island, introduced into the House of Representatives H.R. 9467. This contains Social Security amendments which will vitally affect a great many persons. A complete analysis of the 40-page bill would be longer than is justified in this space. The following is for the purpose of advising the Kansas Medical Society of a particularly significant legislative action that is currently before the Congress of the United States.

Should this bill pass, Social Security taxes will be paid on income up to $6,000 each year, and by 1974 will raise to 7⅛ per cent the tax for the self-employed and to 4¾ per cent the tax to be paid by the employer and by the employee, making a total of 9½ per cent for wage earners.

Under the benefits, it offers 60 days of hospitalization in any 12-month period, plus surgical benefits, and an additional 60 days of nursing home care to all Social Security beneficiaries 65 years of age and older and the same benefits to their survivors and dependents.

The bill says any individual may "freely select the surgeon of his choice, provided that the surgeon is certified by the American Board of Surgery or is a member of the American College of Surgeons except that such certification shall not be required in cases of emergency where the life of the patient would be endangered by any delay, or in such other cases where such certification is not practicable, and except that, in the case of oral surgery, such individual may select a duly licensed dentist."

The bill also says that the Secretary of Health, Education, and Welfare may enter into agreements with individual physicians on the basis of contracts and fee schedules or with an association. He may also make contracts with voluntary non-profit insurance companies.

In brief, this represents socialized medicine for a very large number of Americans. Dr. David E. Allman, president of the American Medical Association, made the following official comment on this proposed legislation and asks all doctors to cooperate in preserving free enterprise in the practice of medicine:

"This proposal is clearly 'socialized medicine' for a segment of the American people. Enactment would permit the federal government to withdraw social security taxes on a compulsory basis from almost the entire working population and use those taxes to reimburse hospitals and physicians for services rendered to all persons eligible to receive old age and survivors benefits. The American Medical Association has repeatedly opposed compulsory health insurance and is unequivocally opposed to this new version."

Test Your Tax I. Q.

Editor's Note. The following series of questions was prepared by the American Institute of Certified Public Accountants in cooperation with the Internal Revenue Service as a method of testing knowledge on the federal income tax law. Answers to the questions will be found on Page 25.

1. During the past year you spent approximately $1,000 for built-in bookcases and wall-to-wall carpeting for your office. Since your lease has only four years to run, you may . . .

(a) Deduct the $1,000 on your 1957 tax return
(b) Amortize the cost over the next four years
(c) Depreciate it over the life of the furnishings

2. When you were transferred to another city, your company gave you a sum of money toward the cost of moving you and your family. For tax purposes you should consider this money as . . .

(a) A gift that is not taxable
(b) Income that is subject to tax with a deduction for only your personal moving expenses
(c) Income that is subject to tax with a deduction for the cost of moving your entire family

3. You have invested in several blue-chip stocks. The dividends received from this investment are exempt up to . . .

(a) $50 whether you or your wife owns the stock
(b) $100 if the stock is held jointly by you and your wife
(c) $100 regardless of who owns the stock, providing you file a joint return with your wife

4. You are *not* permitted to deduct as contributions your donations to which of the following organizations . . .

(a) Charitable societies
(b) Educational institutions
(c) Political parties

5. Your daughter, who was hospitalized for several weeks in the earlier part of 1957, was married in November. If she files a joint return with her husband, you may . . .

(a) Not claim her as a dependent but you may deduct her medical expenses
(b) Claim her as a dependent and deduct her medical expenses
(c) Not claim her as a dependent and you may not deduct her medical expenses

6. You filled very few inside straights during the past few months and lost approximately $300 to the members of your Thursday night poker club. You should . . .

(a) Deduct the loss in computing adjusted gross income
(b) Subtract the loss from adjusted gross income
(c) Give up poker and start watching television on Thursday nights

7. Last October your car skidded on a wet road and grazed a telephone pole. The damage was not covered by insurance and it cost you $100 to have the car repaired. To claim a casualty deduction . . .

(a) You must have the damage repaired within 30 days of the accident

(b) You may simply deduct the amount of the repair bill
(c) You must prove that you were using the car in your work at the time of the accident

8. Which of the following may you *not* consider as a deductible business expense . . .

(a) A subscription to (insert the name of your trade publication)
(b) Commutation fees
(c) Costs of attending the (insert the name of a trade) convention

9. While playing hide-and-seek in your backyard, the neighbor's children trampled and killed several of your more expensive bushes. The cost of replacing this shrubbery . . .

(a) May be deducted if it does not exceed the original cost of the bushes
(b) May be deducted only if the parents of the children refuse to pay damages
(c) May not be deducted under any circumstances

10. Your 16-year-old son works during the summer for you in your unincorporated business, and you pay him a weekly salary. Since he is a full-time employee, he is . . .

(a) Required to pay social security
(b) Not subject to social security
(c) Permitted to decide whether he does or does not want social security coverage

11. Last year you gave your church a small piece of property for which you had paid $500 some time ago. Its value at the time of the gift was $1,500. As a result . . .

(a) You may claim a tax deduction of $1,500
(b) You must pay a capital gains tax on the $1,000 increase
(c) You may claim a tax deduction of $500

12. There were a few leaks in the shingle roof of your office building; so you constructed a new tile roof. You should . . .

(a) Consider this as a repair bill and deduct the entire amount as a business expense on your 1957 return
(b) Regard this as a capital improvement and depreciate the cost over a period of years
(c) Add the cost of the repair to the value of the property

13. After you have filed your personal 1957 tax return, the Government is allowed to check your return and bill you for additional tax. The period of time in which this may be done ends . . .

(a) On the day you file your 1958 return
(b) Two years after you file your 1957 return
(c) Three years from the due date of your 1957 return

14. On the advice of a friend, you engage a CPA to prepare your 1957 tax return. The fee he charges for this service is . . .

(a) Not deductible since it is a personal expense
(b) Not deductible if you are entitled to a refund
(c) Deductible in full

Mid-West Cancer Conference

The 10th annual Mid-West Cancer Conference will be held at the Hotel Broadview, Wichita, March 13 and 14, 1958, according to an announcement made recently by the American Cancer Society, Kansas Division. The Medical and Scientific Committee, with Dr. D. Cramer Reed of Wichita as chairman, has been hard at work on the program for a full year and promises a session that will be interesting and helpful to all Kansas physicians.

The complete program will be announced as soon as possible. A partial list of speakers and their subjects follows:

Dr. Arthur Upton, research pathologist, Oak Ridge National Laboratory, "The Role of Hormonal Factors in the Pathogenesis of Leukemia" and "Hazards of Ionizing Radiation of Practical Concern to Physicians."

Dr. J. A. del Regato, director, Penrose Cancer Hospital, Colorado Springs, "The Treatment of Hodgkin's Disease, Lymphosarcoma, and Leukemia."

Dr. George Moore, director, Roswell Park Memorial Institute, Buffalo, "The Use of Anti-Cancer Agents in Conjunction with Surgery," and "The Treatment of Advanced Breast Carcinoma with TSPA."

Dr. George Crile, Department of Surgery, Cleveland Clinic, "The Role of Endocrine Glands in the Production and Treatment of Cancer."

Speakers for whom subjects have not been announced are: Dr. Joseph W. Beard, Duke University School of Medicine; Dr. Maxwell M. Wintrobe, University of Utah College of Medicine; Dr. G. Burroughs Mider, associate director in charge of research, National Cancer Institute, and Dr. David Karnofsky, Memorial Hospital, New York City.

The motion picture library of the American Medical Association reports that more than 500 film shipments were made during the month of October, 1957. All were supplied to members of the medical profession.

Maternal Death Study—Case History

Editor's Note. This is the first of a series of case reports prepared by the Committee on Maternal Welfare to illustrate the type of study made in each instance of maternal death in Kansas. The committee also records its decisions as to the most probable cause of death, the preventability or unpreventability of the death, and the major contributing factors.

The patient was a 33-year-old multipara who died in a moderately large, fairly well equipped hospital with a certificate diagnosis of rupture of the uterus. A stillborn male child of term size was delivered by laparotomy prior to death of the mother. No autopsy was performed and the case was not discussed by the hospital staff.

Medical history revealed no significant illnesses, and previous deliveries were normal. This pregnancy was apparently normal for the first five months as the patient did not report for care until that time. Subsequent pre-natal records were minimal in character and pre-natal visits were irregular. A complete blood count was performed at the first visit but typing was not done at that time. Diet was thought to be excellent.

The latter weeks of pregnancy were apparently attended by considerable subjective discomfort for the patient. The physician's position was complicated by a close personal friendship with the patient and her husband. He was prevailed upon to attempt induction of labor about two weeks prior to the estimated date of confinement. Quinine and castor oil were used unsuccessfully. Two weeks later, as the result of further insistence, a second induction was initiated.

The patient was admitted to the hospital at noon the day before her expected date. On admission, temperature, pulse, and respiration were normal. Examination revealed the fetus in cephalic presentation with the head floating. The cervix was closed, and there was only slight if any effacement. The fetus was thought to be of mature size, and heart tones were good at 136. There were no contractions. Blood pressure was 108/70.

One ampule of pitocin in one liter of 5 per cent dextrose in water was started intravenously and given at varying rates, a total of ten hours being required for completion. Contractions varied in intensity and rate but at the end of the administration, the cervix was not effaced and the dilatation was two centimeters. One and one-half hours after the fluids were terminated, contractions continuing, the patient had a generalized convulsive seizure immediately followed by severe shock, and she did not regain consciousness again. One and one-half hours after the convulsion, blood was started. One hour later, following obstetrical consultation, laparotomy was performed and the uterus was found to be ruptured, the dead baby being free in the abdomen. Oxygen was administered, transfusions were continued, further consultations were obtained, but the patient did not respond and expired about four hours later.

Committee Action

Following presentation of the case by the physician carrying out the study, discussion by the Committee on Maternal Welfare emphasized the fact that attempted induction of labor, with a potent oxytocic in the presence of an unripe cervix without obstetric indication violated a basic principle of obstetric management. The delay in diagnosis and institution of therapy for shock and the delay in accomplishing the laparotomy were considered to be major factors in the fatal outcome.

CLASSIFICATION: DIRECT OBSTETRIC DEATH; PREVENTABLE; ERROR OF ATTENDING PHYSICIAN.

Clinicopathological Conference

Jaundice, Paronychiae, and Generalized Purulent Skin Lesions

Case Presentation

The patient for discussion today was a 25-month-old Negro boy who was first admitted to this hospital on March 7, 1956, with the complaint of "liver trouble" of two months' duration.

He had apparently been well until December, 1955, when his fingernails began to split and fall off, and pruritic, scabbing, purulent lesions developed on his scalp. These lesions gradually spread over the rest of his body. In January, 1956, he became extremely lethargic, his sclerae became noticeably yellow, his urine was dark, and he had eight to ten light yellow, watery or cheesy stools daily.

The family lived in a neighborhood with outside toilets which were frequently infested with rats. They used city water and dairy milk. In June, 1955, he had gone to Texas to visit cousins who had lesions on their scalps which were said to be similar to those which the patient had developed. He had had no contact with jaundiced persons.

The boy was well developed but poorly nourished, weighing only 8.9 Kg. (19½ pounds). He was alert, irritable, and obviously jaundiced. There were numerous crusted lesions on the scalp, axillae and groins, and a brown punctate rash on the arms, legs and soles of his feet. Crusting, exfoliating lesions had almost destroyed his fingernails, and there were several large paronychial vesicles which were filled with a purulent material. The sclerae were icteric. The pupils were round and equal and reacted to light. The ear canals were filled with a purulent exudate and bloody crusts, and both pinnae were covered with vesicles and pustules. The lips were cracked and bleeding; the tongue was moist, and the gums were dark and bled easily. Numerous pinpoint gray-brown spots were noted inside the upper lip, and the oral mucosa was icteric. The lung fields were clear, and the heart tones were normal. The liver was palpable 5 cm. below the right costal margin; the spleen, 2 cm. below the left costal margin. No other organs or masses were palpated.

On admission the red count was 5,600,000 with 2.6 per cent reticulocytes and 6.5 gm. of hemoglobin. There was anisocytosis, hypochromia, and microcytosis. The white count was 12,000 with 73 per cent polymorphonuclears, 19 per cent lymphocytes, and 1 per cent eosinophiles. The urine was positive for bile and negative for cytomegalic inclusion bodies. The VDRL was non-reactive. The heterophile titer was 1:14. Leptospira studies, febrile agglutinins, and blood smears for malarial parasites were all negative. The blood sugar was 83 mg. per cent; urea nitrogen, 10.5 mg. per cent; sodium, 138 mEq/L; potassium, 5.0 mEq; carbon dioxide, 24 mEq; chlorides, 108 mEq. The total serum bilirubin was 10.2 mg. per cent with 6.6 mg. per cent direct bilirubin. The alkaline phosphatase was 31.3 millimol units; cephalin cholesterol, negative; thymol turbidity, 21 units; serum albumin, 4.6 gm. per cent; globulin, 2.4 gm. per cent; and the serum cholesterol was 412 mg. per cent with 28 per cent esters. The stools were negative for ova and parasites. Cultures of the nose and throat grew out pneumococci, hemolytic streptococci and Pseudomonas. Cultures from the axillae yielded Proteus and a moderate growth of Candida albicans. Three blood cultures were negative at ten days. The prothrombin time was less than 10 per cent of normal. The glucose tolerance curve was flat. Serum electrophoresis showed a relative decrease in albumin and normal gamma globulin. A sickle cell preparation and Coomb's test as well as tuberculin and histoplasmin skin tests gave negative results. The serum iron was 76.5 gamma per cent, and the serum transaminase was 84.6 GOT units. The lupus erythematosis cell preparation was negative.

The infant was treated with bacitracin ointment, erythromycin by mouth, and parenteral menadione sodium bisulfite (Hykinone). He was given a high protein, high calorie diet, but he did not improve. A liver biopsy was done, and the extrahepatic ducts were explored and found to be normal. When ascites developed, cortisone was added to his regimen, but it was gradually discontinued. During most of this hospital stay he was afebrile, with only an occasional elevation in temperature to 103 degrees. He was discharged on June 9, 1956, unimproved, but weighing 13 Kg. (28½ pounds).

On July 29, 1956, he was readmitted with anorexia, fatigue, and loss of weight. His physical findings were essentially the same as on the previous admission. He weighed 11 Kg. (24 pounds), was marked-

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Edited by Jesse D. Rising, M.D., and Mahlon Delp. M.D., from recordings of the conference participated in by the departments of medicine, pediatrics, surgery, radiology, and pathology of the University of Kansas Medical Center, as well as by the third and fourth year classes of medical students.

ly jaundiced, and the skin lesions were more severe. Laboratory findings at this time showed albuminuria, but were otherwise unchanged.

He was started on a cholesterol-free diet, bacitracin ointment, and vitamins, iron, and erythromycin by mouth. He was discharged on August 4, 1956, and followed afterward in the outpatient clinic, where he was treated with permanganate soaks, gentian violet, sodium liothyronine (Cytomel), triple sulfonamides, and oxytetracycline hydrochloride by mouth.

He did not return to the clinic after August, but on December 6, 1956, he was brought to the emergency room, bleeding from the nose, mouth and bowels, and having vomited blood. He was readmitted to the hospital at that time.

He was extremely jaundiced, poorly nourished, alert and apprehensive. His skin was covered with dried pustular lesions, and the tips of the fingers and toes were swollen and bloody with split and missing nails. The skin of the axillae and groins was excoriated. The findings in the abdomen were unchanged.

The white count was 53,000 with 71 per cent polymorphonuclears, 29 per cent lymphocytes, and 1 per cent monocytes. The hemoglobin was 2.6 gm. The platelet count was 730,000. The serum albumin was 2.66 mg. per cent; globulin, 1.44 mg. per cent. The prothrombin time was over five minutes.

The child's condition became progressively worse, and shortly after admission he suddenly died during an attempt to insert an intravenous cannula.

Dr. Mahlon Delp (moderator): Are there any questions for Dr. Cherry?

Alfred Scherer (fourth year medical student):* Did he have any bleeding before his first admission?

Dr. Arthur Cherry (resident in pediatrics): No, he did not.

Mr. Scherer: Was there a family history of jaundice?

Dr. Cherry: Not that we could determine.

Warren E. Roberts (fourth year medical student): Had this child ever received intravenous or intramuscular medications before his first admission?

Dr. Cherry: He had received only oral and local medications.

Erich Ryll (fourth year medical student): Had he been exposed to any toxins?

Dr. Cherry: He may have been given a mixture of turpentine and water, but there is no definite record of this.

Charles L. Schroff (fourth year medical student): What were the serum iron and transaminase values?

Dr. Cherry: Only the one transaminase was done; a second serum iron level was 75.1 gamma per cent.

Mr. Ryll: Were any other serum cholesterol values obtained?

Dr. Delp: One was 660 mg. per cent; another, 700 mg. per cent; and a third, 1120 mg. per cent.

Mr. Scherer: Was he on a milk diet before his first admission?

Dr. Cherry: He was breast fed until he was nine months old; then he was given table foods. He refused homogenized milk, and he was given no vitamins. He seemed to have been allergic to orange juice, and it is recorded elsewhere that he had been fed mostly eggs and toast during his early months.

Mr. Searle: Was the glucose tolerance curve high or low?

Dr. Cherry: The fasting value was 90 mg. per cent, and the others were 96 mg. per cent, 92 mg. per cent, and 96 mg. per cent. There was no sugar in any of the urine specimens.

Mr. Scherer: What was the platelet count?

Dr. Cherry: The platelet count was 667,000 on the first admission.

Mr. Schroff: What was the prothrombin response from the vitamin K he received on his first admission?

Dr. Cherry: The first two prothrombin times were less than 10 per cent of normal. On April 4 it was 82 per cent of normal, and on May 28 it was 83 per cent of normal.

Mr. Scherer: What was his temperature on admission?

Dr. Cherry: It was normal.

Mr. Searle: I assume a T-tube was used after the biliary exploration; was there any drainage?

Dr. Cherry: There was drainage of yellow fluid.

Dr. Delp: How long was the T-tube left in?

Dr. Cherry: Only a short time.

Mr. Scherer: What did the urinalysis show besides the albuminuria on the second admission?

Dr. Cherry: The voided specimen was neutral with a specific gravity of 1.002. There was a heavy trace of albumin, but no sugar. There were rare white blood cells per low power field, some mucous and positive bile.

Mr. Ryll: What were the results of the skin and fingernail cultures?

Dr. Cherry: The skin and fingernail cultures showed nonhemolytic staphylococcus aureus, Pseudomonas, Candida albicans, and Proteus.

Mr. Searle: What were the alkaline phosphatase, cephalin cholesterol, and thymol turbidity values on the first admission?

Dr. Cherry: The alkaline phosphatase was 31 units, initially, and the next determination was 50 units. The cephalin cholesterol was 1 plus, and the thymol turbidity was 37 units. The next hepatogram showed

* Though a medical student in March, 1957, when this conference occurred, he, like the others referred to as students, received the M.D. degree in June, 1957.

an alkaline phosphatase of 66 units; the cephalin cholesterol was negative, and the thymol turbidity was 24 units. A repeat test was similar.

Mr. Ryll: Was the mother subject to fever blisters?

Dr. Cherry: I do not know.

Mr. Searle: Will you describe the liver on the third admission?

Dr. Cherry: It was palpable three centimeters below the right costal margin, and it was firm but not tender.

Mr. Schroff: Did the itching begin before the skin lesions were noted? And when did the mother first notice that the urine was dark?

Dr. Cherry: The skin lesions were noted before the itching began. The urine was first noticed to be dark yellow in January, 1956, before the mother first saw a physician.

Dr. Delp: May we see the electrocardiogram, please?

Mr. Roberts: This tracing was taken in May, 1956 (Figure 1). There is a normal sinus rhythm, and the rate is 130. I interpret this as a normal tracing with a sinus tachycardia.

Dr. Delp: Now may we have the x-rays, please?

Mr. Scherer: The cholangiogram taken on April 20, 1956 (Figure 2), shows the T-tube in place. The gallbladder fills well, and the dye is seen in the small intestine. I do not see any dye in the hepatic radicles.

The admission chest film shows no bony abnormalities; the lung fields are clear; and the heart is of normal size for this age. The liver is enlarged, and the shadow of the spleen is visible. There is diffuse haziness over the abdomen suggesting ascites. I cannot see the psoas shadows. The bowel pattern seems normal.

The films of the skull taken on the first admission

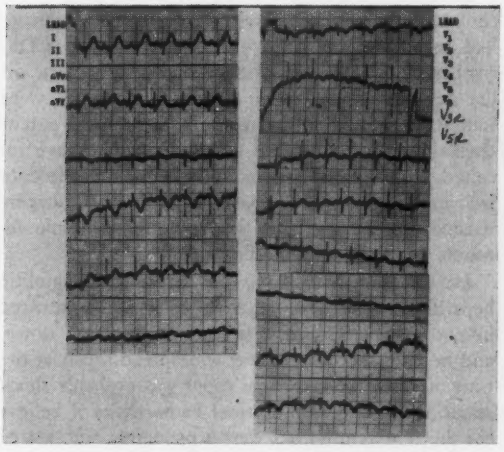

Figure 1. Electrocardiogram taken in May, 1956.

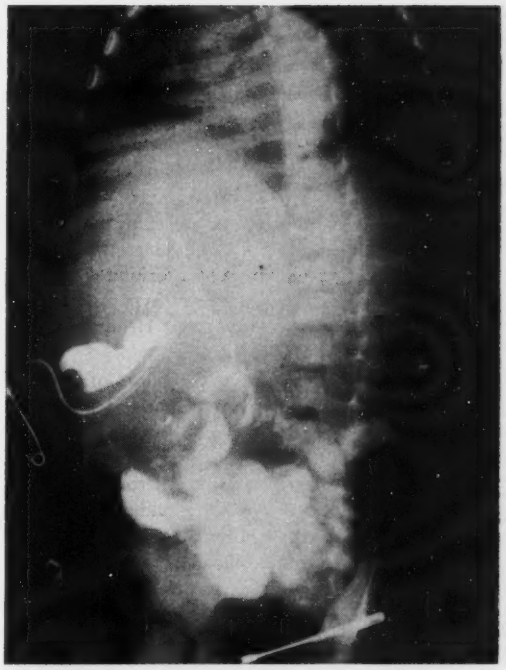

Figure 2. Cholangiogram taken on April 20, 1956.

show no punched-out areas or abnormal calcifications. The sella is not enlarged. There are no bony abnormalities. I interpret these as normal skull films.

Dr. Delp: Dr. Youngstrom, do you have any comments?

Dr. Karl Youngstrom (radiologist): In the cholangiogram the hepatic radicles did not fill as well as they should.

Dr. Delp: Mr. Schroff, may we have your differential diagnosis and cause of death, please?

Differential Diagnosis

Mr. Schroff: The case for discussion today is that of a 25-month-old Negro boy who was apparently well until three months before admission, when he developed pruritic skin and nail lesions. In January, 1956, his eyes were yellow, his urine was dark, and he began to have numerous light yellow, watery or cheesy stools.

He was a well developed, poorly nourished, jaundiced child with widespread purulent skin and nail lesions. He had hepatomegaly and splenomegaly. Laboratory tests showed a mild leukocytosis, hypochromic anemia, and markedly abnormal liver-function studies which indicated both biliary obstruction and parenchymal cell damage. He was treated with antibiotics and diet, but, except for a good response to vitamin K therapy, he did not improve. His extrahepatic ducts

were explored and found to be normal. We do not know what the liver biopsy showed. The child developed ascites and fever and was not improved at the time of discharge. When he was seen four months later his condition had deteriorated noticeably, and he was treated with antibiotics, iron, and diet. He was again discharged to be followed as an outpatient.

The last admission was in December, 1956, approximately one year after the onset of his illness. He was bleeding from the nose and gastrointestinal tract. His condition had further deteriorated, and he had severe anemia, leukocytosis, hypoproteinemia, and a prolonged prothrombin time. He died shortly after admission.

I shall base my discussion on the causes of jaundice in infancy. These can be grouped under two main headings: obstructive and non-obstructive. In this case the xanthomata, pruritus, high direct bilirubin, high alkaline phosphatase and high serum cholesterol are suggestive of obstructive-type jaundice. The length of the clinical course is against this, and the hepatomegaly, elevated serum iron, and transaminase suggest hepatocellular damage. Furthermore, we know the extrahepatic ducts were found to be normal. Intrahepatic atresia is ruled out because of the length of the clinical course and the drainage of bile from the T-tube; but intrahepatic obstruction, caused by some type of cirrhosis, is not so easily eliminated.

Of the non-obstructive causes, I will consider hemolysis and parenchymal cell destruction. Our patient may have been hemolyzing, but there was no spherocytosis, and a sickle cell preparation was negative. A primary hemolytic process alone cannot account for the clinical and laboratory findings in this case. There are many causes of liver cell destruction such as toxins, pyrogens, parasites, spirochetes, and fungi. None of these seem to warrant serious consideration except, perhaps, syphilis, leptospirosis, and amebiasis, and these can be ruled out by the negative blood and stool studies.

I shall now consider viral agents which cause jaundice. Cytomegalic inclusion disease is usually seen in the neonatal period. In older infants it is frequently associated with pneumonitis and enterocolitis. In the absence of these findings and in the absence of inclusion bodies in the urine sediment, I shall rule out this disease. Infectious mononucleosis may cause jaundice, but this is usually a mild disease associated with posterior auricular lymphadenopathy and a high heterophile titer. I think we can eliminate it as a possible diagnosis.

Finally, I shall consider viral hepatitis. I shall make no attempt to determine the etiology because infectious hepatitis and homologous serum hepatitis cannot be differentiated clinically. It has long been considered that viral hepatitis in children is a mild disease, but it has been found that hepatitis can occur in three general syndromes: the largest group follow a relatively benign course; a few develop fulminant hepatitis in which death occurs within ten days; and a third group has a progressive hepatitis. The last type of disease is believed to be an unrelenting process which is usually accompanied by portal hypertension and results in death within two to nine months. There are numerous reports in the literature of progressive hepatitis in children simulating biliary atresia. Clinically, there are many pictures of obstructive jaundice with acholic stools, and, were it not for the elevated serum iron and transaminase tests now being recorded, hepatitis might not even be suspected.

The onset is usually in the neonatal period, but it may occur later. The child is active and alert, has a low-grade fever, and generalized lymphadenopathy. The liver and spleen are both enlarged. The serum bilirubin is elevated to about 10 mg. per cent, especially in the direct component. Urinary urobilinogen is usually absent, and fecal urobilinogen is absent or low. Thymol turbidity and cephalin cholesterol flocculation may be normal or elevated. Alkaline phosphatase and total cholesterol are frequently elevated. An abdominal exploration and a liver biopsy are necessary for a definite diagnosis. The condition progresses to cirrhosis in a few weeks, and it may be difficult to distinguish from Laennec's cirrhosis or portal cirrhosis. It has been called a variety of names including Hanot's cirrhosis, primary biliary cirrhosis, and xanthomatous cirrhosis. It is probably best termed cholangiolitic biliary cirrhosis.

The clinical picture is one of progressive deterioration with persistent jaundice. Circulatory changes occur which result in portal hypertension, hepatomegaly, splenomegaly, ascites, and esophageal varices. Death results from liver failure and hemorrhage.

In summary, I believe this patient had progressive cholangiolitic hepatitis of viral etiology. He may have had an unrecognized active infection as early as December, 1955. The infection progressed so that, on the first admission, the liver function studies were indicative mainly of obstruction with minimal parenchymal damage. The low hemoglobin may have resulted from gastrointestinal bleeding or inadequate iron in his diet. He may have had mild portal hypertension, but I believe the low prothrombin time accounts for most of the bleeding.

He continued to have an active cholangiolitic hepatitis with progressive cirrhosis which eventuated in portal hypertension. His condition became worse, and he had liver failure and widespread sepsis at the time of death. His terminal event was probably shock resulting from gastrointestinal hemorrhage. I believe the pustular skin lesions were a pyoderma and related to his primary disease only because they were ag-

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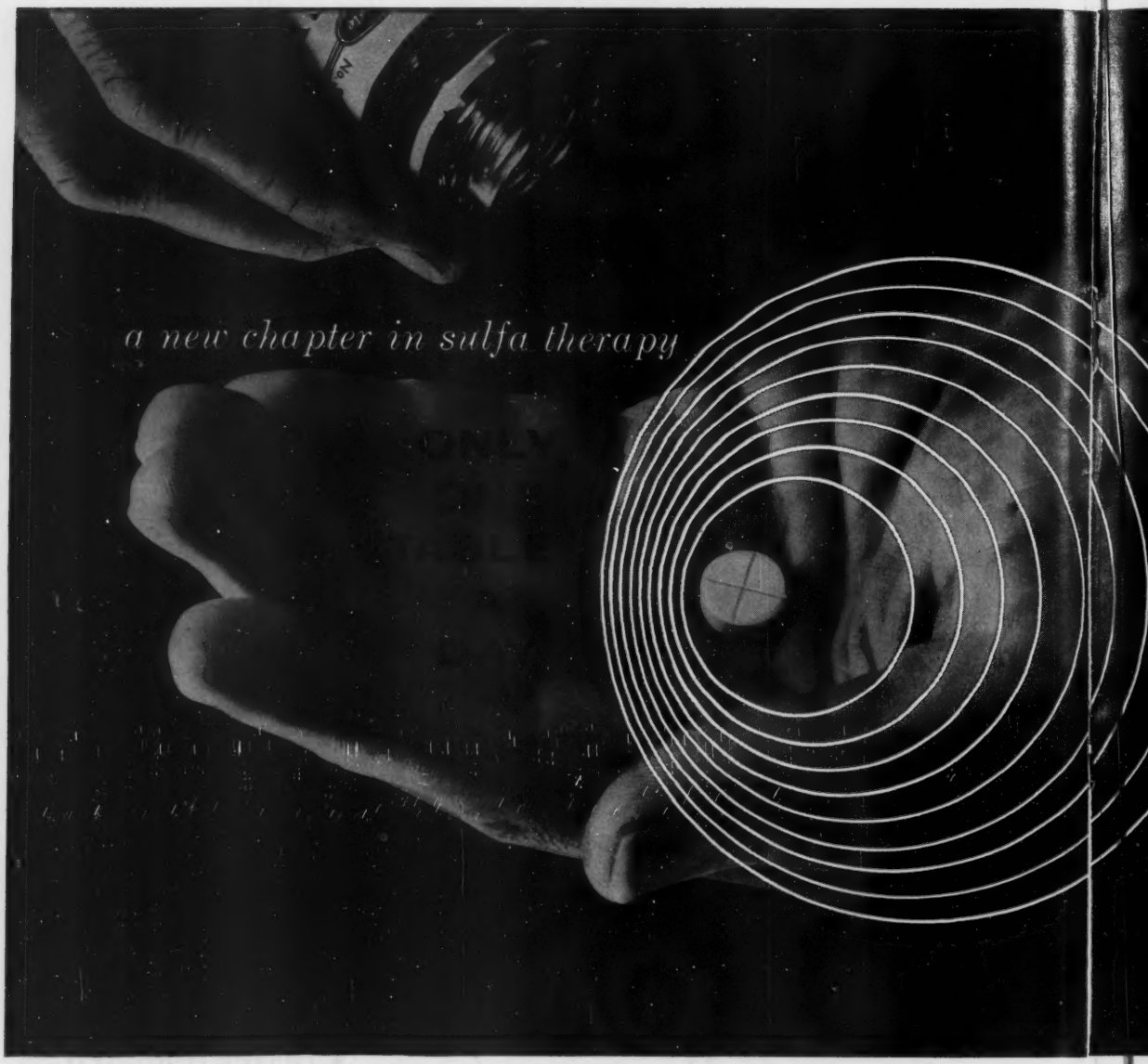

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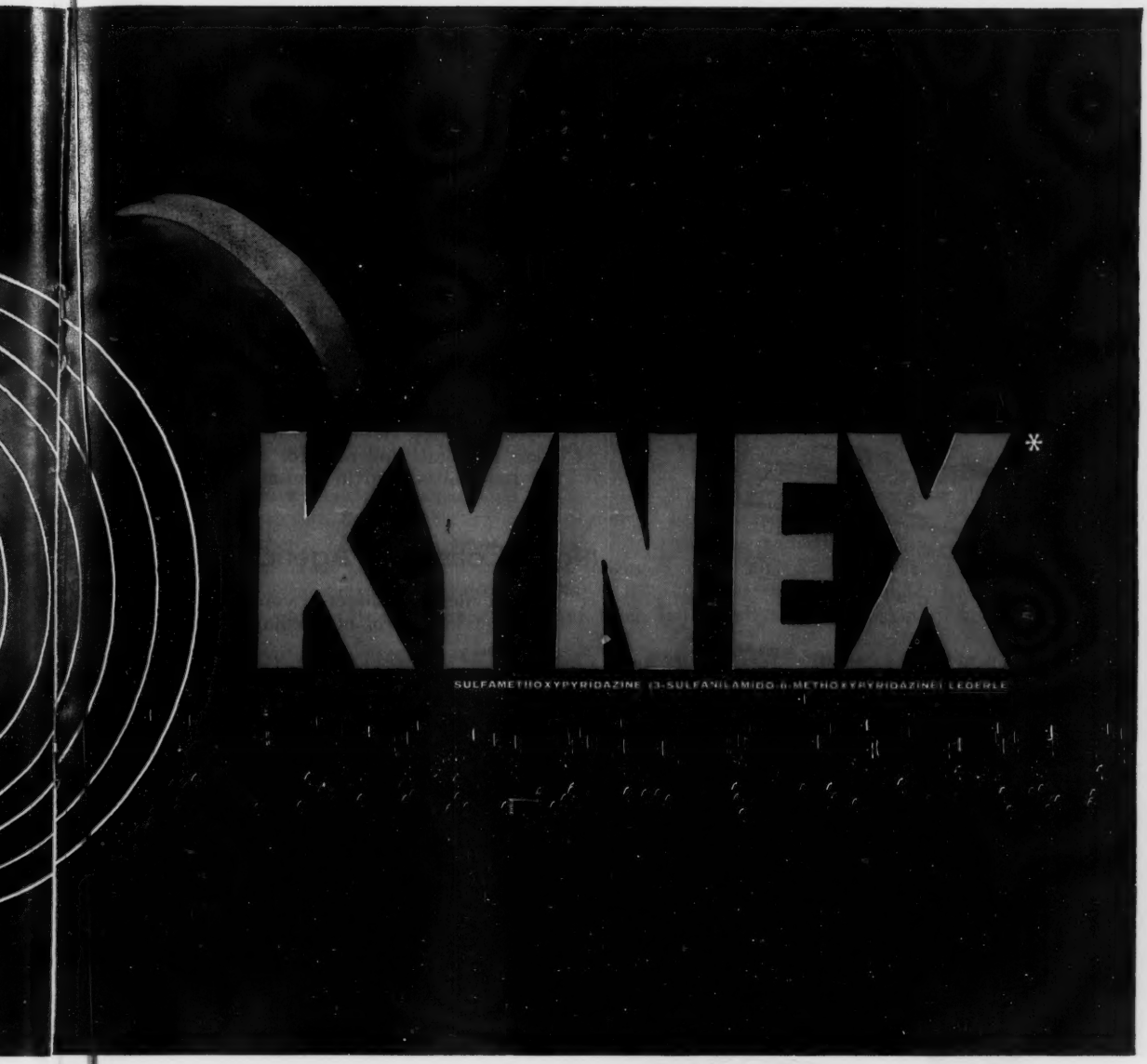
1. Nichols, R. L. and Finland, M.: *J. Clin. Med.* 49:410, 1957.



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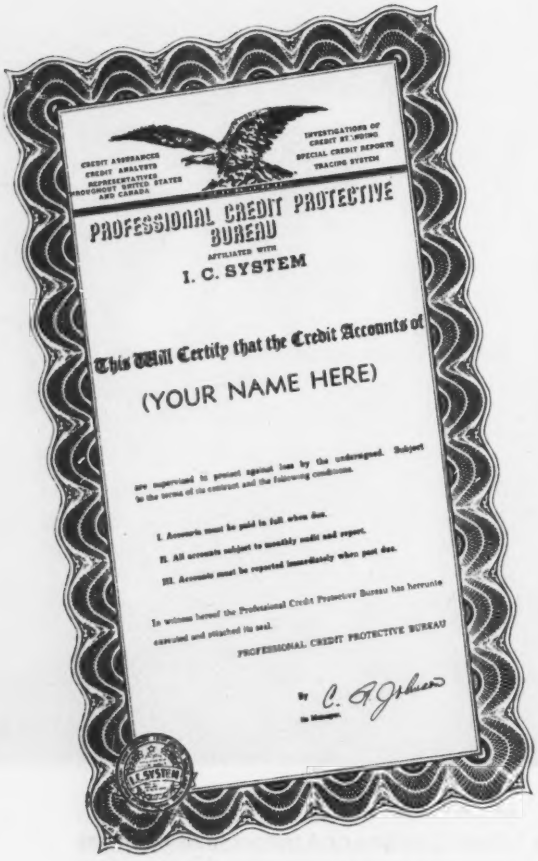
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gravated by the pruritus (secondary to jaundice) and scratching. His nutritional status and general debilitation may have been the cause of the poor response of these lesions to therapy.

Clinical Discussion

Dr. Delp: Thank you. What is your diagnosis, Mr. Scherer?

Mr. Scherer: Progressive cholangiolitic hepatitis.

Dr. Delp: Mr. Searle?

Mr. Searle: The same.

Dr. Delp: Mr. Scherer, what is your explanation of the skin lesions, especially those on the fingers and toes?

Mr. Scherer: They could have started as a mycotic infection of the fingernails.

Dr. Delp: Mr. Searle?

Mr. Searle: He may have had a fungal infection. I cannot rule out vitamin C deficiency, although this may not have played an important role.

Dr. Delp: Mr. Roberts, what is your explanation?

Mr. Roberts: I believe fungi were cultured.

Dr. Delp: There was only one time when a fungus was cultured, and that was from the axillae. Mr. Ryll, why did you ask whether the mother had fever blisters?

Mr. Ryll: Several cases have been reported in the literature of a herpetic infection involving the liver. It is believed that the infant acquires the disease from fever blisters on the maternal vulva. However, most of those cases terminate more quickly than ours did.

Dr. Delp: Mr. Searle, if we should accept the diagnosis of cholangiolitic hepatitis, why didn't this child recover? And why was this disease progressive?

Mr. Searle: I think he had progressive viral infections.

Dr. Delp: Do you believe that he had a viremia during the entire year?

Mr. Searle: No, because the virus was not found after the first two weeks.

Dr. Delp: Mr. Roberts, is it true that the virus must be found within the first two weeks or it is not found at all?

Mr. Roberts: In the acute stage, the virus is found in the blood stream, then the viremia subsides.

Dr. Delp: Why was this disease progressive?

Mr. Ryll: We cannot rule out the fact that the infant may have been infected transplacentally. The mother may have been a silent carrier without any symptoms herself. Cases of homologous serum jaundice have been reported in which this has occurred, and the disease continued insidiously and progressively.

Dr. Delp: The child was symptom free until about 18 months of age, was he not?

Mr. Ryll: That is how it was reported.

Dr. Delp: Do you think he might have had a viremia since uterine life?

Mr. Ryll: It is possible.

Dr. Delp: Do you think he had a viremia during this whole period of time?

Mr. Ryll: No, only at the time of the acute disease; but one cannot rule out infections at various times.

Dr. Delp: Mr. Schroff, why was his disease so relentless? Did he have a constant viremia?

Mr. Schroff: No, I did not think so, but he probably had an active hepatitis most of the time. The liver has tremendous regenerative powers despite a continuing destructive process.

Dr. Delp: Mr. Scherer, why was this child jaundiced?

Mr. Scherer: Because the permeability of the cholangioles was altered allowing diffusion of bile through the cholangioles back into the blood stream.

Dr. Delp: Was this an obstructive jaundice, Mr. Schroff?

Mr. Schroff: Physiologically, yes; anatomically, no. There was no actual obstruction in the biliary system, but there was no outflow of bile, because it went back into the blood stream.

Dr. Delp: Then you would call this a "lower lobule hepatosis"? The child came into the hospital and died rather precipitously; why was that, Mr. Scherer?

Mr. Scherer: I think he was in shock, secondary to hemorrhage.

Mr. Ryll: Such patients die during relatively minor procedures. Our patient died when a cannula was being inserted into a vein, and this procedure may have precipitated his death.

Dr. Delp: Mr. Schroff?

Mr. Schroff: I think the prothrombin response on his final admission was low, and he may have had esophageal varices.

Dr. Delp: Esophageal varices from portal hypertension?

Mr. Schroff: Yes.

Dr. Delp: Do you think he had portal obstruction?

Mr. Schroff: Yes, I do.

Dr. Delp: Do you think this is a part of the disease entity?

Mr. Schroff: Yes. There is portal obstruction when the disease progresses to cirrhosis.

Dr. Delp: Mr. Scherer, do you have an explanation for the unusual cholesterol values?

Mr. Scherer: He was reabsorbing the cholesterol, and the cholic acid in his blood was high. The entire breakdown of cholesterol is cholic acid, and it has been postulated that the equilibrium is reversed, resulting in an increased cholesterol synthesis.

Dr. Delp: Mr. Schroff?

Mr. Schroff: This may have been a lipoid storage disease, but I do not consider it likely because there was no other evidence.

Dr. Delp: Do you think the lesions on the fingers were xanthomata?

Mr. Schroff: I do not know.

Dr. Delp: Mr. Schroff, do you think the pathologist will find obstruction in the liver?

Mr. Schroff: I think he will find Laennec's cirrhosis. I do not expect to see many normal hepatic cells or bile plugs except for some bile in the smaller ducts.

Dr. Delp: What do you think about this problem, Dr. Miller?

Dr. Herbert C. Miller (pediatrician): I believe that the lesions on the fingers are somehow related to the disease. I know of no fungus that does such extensive damage in this climate.

Dr. Delp: Do you think the child had a viremia from the onset of his disease in November, 1955, to December, 1956, when he died?

Dr. C. Arden Miller (pediatrician): No, I doubt it.

Dr. Delp: Can you explain why the disease was so relentless?

Dr. Miller: No, I cannot.

Dr. Delp: Do you have any comments about the fingers?

Dr. Miller: I believe they were xanthomata.

Dr. Delp: Mr. Searle, do you agree that an exploratory laparotomy on this child was justified?

Mr. Searle: The liver biopsy was the only way to make a positive diagnosis, and a punch biopsy on a child is a risky procedure.

Dr. Delp: Dr. Manning, what do you think about the concept of hepatosis?

Dr. Robert Manning (resident in medicine): Dr. Sherlock[5] has reported cases of viral agents producing an unusual lesion in children. The liver has an abnormal response which pathologically shows formation of alveolar-like structures instead of liver cords in response to the injury. It would be difficult in this case to differentiate primary intrahepatic cholestasis from primary intrahepatic biliary atresia.

Dr. Delp: What do you think the biliary tract will show on the pathologist's report?

Dr. Manning: It will be patent extrahepatically and obstructed intrahepatically.

Dr. Delp: Dr. Andrews, what do you think about this?

Dr. Charles E. Andrews (internist): I suspect there will be intrahepatic obstruction.

Dr. Delp: Dr. Rankin, do you have any comments?

Dr. Thomas J. Rankin (internist): I feel certain that this child had intrahepatic obstructive disease of an infiltrative character, and the case for infection is good. I believe, however, that the lipoid storage diseases should have been more extensively considered in the differential diagnosis.

Dr. Delp: Dr. Weber, do you think this child had a persisting viremia?

Dr. Robert Weber (internist): Yes, I do.

Dr. Delp: Does anyone have any further ideas about the fingers?

Dr. Rankin: In all of the lipoid storage diseases there is a low resistance to infection. This is also true with xanthomatous biliary cirrhosis secondary to infection. In retrospect, I think this child could have developed this infection from interference with nutritional resistance to infection. I am intrigued with the fact that the fingernails were splitting before there was any other evidence of trouble, and for this reason I favor a metabolic disease rather than infection.

Dr. Delp: When I first saw this child he was itching and scratching continuously, and secondary pyoderma developed in the fingernails and on his toes. The idea of the cholesterol and lipid disturbance entering in is good, and is probably the explanation. There is a recent report[1] of a patient with acrodermatitis continua (Hallopeau) which reminds me of our patient, but I am not entirely familiar with that disease entity.

Dr. Frenkel, may we now have the pathological discussion?

Pathological Report

Dr. Jacob K. Frenkel (pathologist): I must admit that I am not familiar with that entity either, and I hope that I will not be asked too many embarrassing questions about it. In the dermatologic literature the most consistent comment about xanthomatosis in childhood was that it required monographic treatment. Crocker's[2] article was perhaps the best. I agree with Dr. Rankin that the various histiocytoses should be considered; however, the spleen, bone marrow, and skull films were normal, and the platelet count was over 700,000. There was no central nervous system involvement. The cholesterol values are essentially normal in individuals with Letterer-Siwe's disease or eosinophilic granuloma, whereas in our patient they were markedly elevated.

The liver was enlarged and weighed 550 gm. There was a slight amount of scarring resembling Hanot's cirrhosis more than postnecrotic scarring or Laennec's cirrhosis (Figure 3). There was some proliferation of bile ducts accompanying the fibrosis. The biopsy showed a few areas where bile was inspissated both within the intrahepatic canaliculi and within the ducts; this had become more marked at the time of the autopsy. A few instances of transition from the bile canaliculi to the intralobular bile ducts were identified. The extrahepatic biliary system was normal in the gross.

The architecture of the liver as a whole was relatively normal and quite unlike that usually found with chronic viral hepatitis and postnecrotic scarring. There were many bile plugs with inflammation around

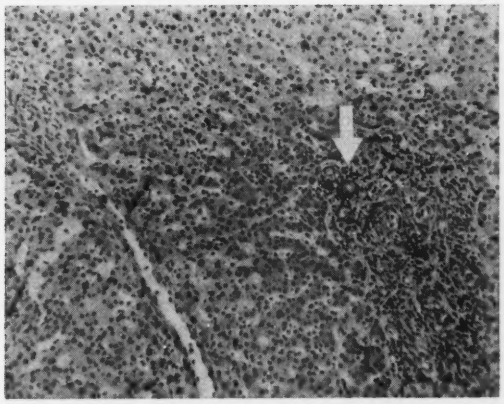

Figure 3. Liver showing parenchymal inflammation and scarring in periportal and central location. Bile plugs are indicated by the arrow. Hematoxylin and eosin X100.

the cholangioles, hence the term cholangiolitic hepatitis, referring to an inflammation of the cholangioles. This was more marked in the biopsy specimen than at the time of autopsy. Degeneration and regeneration of the liver cells persisted, the latter leading to the formation of cells with six or eight nuclei. These findings are perhaps in keeping with long continued viremia or with continued destruction due to the chronic intrahepatic inflammatory process. I cannot distinguish between the two, but tend to favor the latter, as I saw no polymorphonuclears.

The appearance of the reticulum in several areas of the liver indicated a collapse of liver tissue with lobule disappearance. The Masson stain for connective tissue indicated that there was relatively little scarring in contrast to the scarring which occurs in adults.

The results of the obstruction, as Mr. Scherer pointed out, are the piling up of the cholic acid and a disturbed equilibrium with an increase in the amount of cholesterol. There were xanthomata in the liver as well as in the skin.

In regard to the skin lesions, there was a good progression of the changes. In the early xanthomas, the epidermis was somewhat elevated and slightly attenuated by cells containing lipids located in the papillary dermis. It would not be possible to distinguish these sections from those of a case of lipoid storage disease. With progressive infiltration of the dermis by these cells, there was pressure atrophy of the overlying epithelium (Figure 4). Secondary infection resulted in vesicles filled with pus located at the dermal-epidermal junction. Desquamation of the stratum corneum led to the formation of a broadening ulcer base. The bacterial flora of these ulcers was altered by chemotherapy. Enterococci, staphylococci, and Proteus were cultured at the time of autopsy. No fungi were found, either by culture or staining.

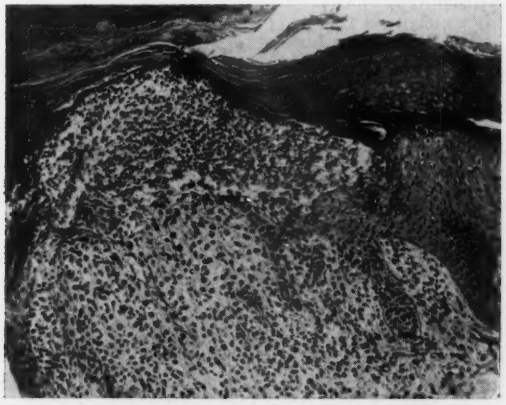

Figure 4. Skin of finger showing focal infiltration of xanthoma cells into papillary dermis with atrophy of the overlying epidermis and the development of a pustule covered by a scab. Periodic acid Schiff stain with hematoxylin. X100.

Figure 5. Photograph of hand showing ulceration of nail beds.

In the nail beds of the fingers and toes the most marked involvement was found, leading to separation of the nail and destruction of the nail bed (Figure 5). The bases of the fingers were relatively free of xanthomas and infection. However, the axillae and groin were markedly involved. I cannot explain why the lipoid containing macrophages accumulated where they did, nor why more were found at the tips of the fingers than at the base.

In summary, I interpret this as a case of cholangiolitic hepatitis. It is not known why the cholangioles appear to be especially vulnerable. Some pathologists doubt that this histological lesion is actually pathognomonic of the disease. We see cholangiolitis in any form of hepatitis, and the amount of cholangiolitis seen at autopsy is quite variable in different

cases of the so-called cholangiolitic hepatitis diagnosed clinically. There appears to be a lack of correlation between the degree of cholangiolitis histologically and the clinical symptoms. I feel that it is proper to retain this designation until there is a better explanation for the process.

An account of the history and section of this case was sent to four pathologists for consultation. Two pathologists from children's hospitals concurred with the diagnosis of cholangiolitic hepatitis, stating that they had seen similar, but less advanced, cases in children. A general pathologist with special interest in the liver interpreted the case as one of congenital biliary atresia, perhaps of incomplete nature and distributed in the larger intrahepatic ducts intervening between the hilar channels and the interlobular ductules. I would be in entire agreement with this diagnosis if there had been no history dating the onset of jaundice, dark urine, and of light stools fairly close to the 24th month of our patient's life. One might also wonder, as did one of the pediatric pathologists, about the cause of so-called biliary "atresia" from a developmental point of view; destruction of bile radicles *in utero* would be more plausible than agenesis. In this case a viral infection with cholangiolitis would be a reasonable possibility.

A fourth pathologist from a children's hospital made the diagnosis of Letterer-Siwe's disease with cholangiolitic hepatitis. Although conceding that the spleen and bone marrow did not suggest this diagnosis, he was impressed by the histiocytosis in the skin, liver, and lymph nodes. This interesting point of view, in my opinion, does not take sufficiently into account the marked hyperlipemia, the xanthomata, and the limitation of reticular cell proliferation to only those lymph nodes proximal to extensively infected skin ulcers.

It is my impression that the patient died from uncontrollable bleeding, secondary to absent or deficient vitamin K intake.

Dr. Delp: Are there any questions of Dr. Frenkel?

Mr. Searle: Can you explain the high platelet count?

Dr. Frenkel: No, I cannot, but it might serve to support the student's contention that the bone marrow was functionally intact. Where there is a bleeding tendency, it is possible to have hyperplasia of the megakaryocytes, but I am not certain of it. Dr. Delp, do you find that the platelet count goes up in bleeding due to low prothrombin?

Dr. Delp: We see it often, but I cannot explain it.

Dr. Frenkel: It is too easy to say that it is a compensatory phenomenon.

Mr. Roberts: Was there anything in the intrahepatic ducts to prevent the dye from going in?

Dr. Frenkel: Many of the ducts were plugged, but most of the plugging was at the margin of the lobules.

Dr. Herbert Miller: Do you intend to convey the impression that this is an unknown entity, or is it of an infectious nature?

Dr. Frenkel: Laboratory tests are not available to prove the diagnosis of infectious hepatitis; the virus can be isolated only in human volunteers. For this reason we can never be certain of any type of viral hepatitis. The concensus is that this may be a complication of "infectious hepatitis."

Dr. Delp: I believe the nail lesions are something new and have not been previously described. The relationship to the high cholesterol in this case makes this something of an original contribution. I have never seen such lesions, but they must be related to the disturbed lipid metabolism. I still have a question: If the child had infectious hepatitis and if viremia was the cause of the hepatitis, did he have a viremia during the year of the illness? I do not know, but I suspect he did have it a good part of the time. I suspect that if volunteers had been injected with his blood, they would have developed infectious hepatitis.

I should point out, however, that it is possible that a hyperreactive state may occur in the liver, much as occurs in the heart that has been insulted with the streptococci and subsequently develops acute rheumatic carditis.

It is becoming increasingly difficult for me to accept the present explanations of cholangiolitic hepatitis. The explanation for the jaundice on the basis of obstruction is not satisfactory. It seems more reasonable to assume that perhaps there is tubular failure within the liver itself. I believe that at the present moment we are in almost the same position that we were a few years ago when we were talking about lower nephron nephrosis.

Summary

Dr. Delp: The history suggested infectious hepatitis. Physical findings of jaundice, pruritis, enlarged liver and, most striking, the bulbous, exudative lesions involving the nails and fingers, were not helpful in the differential diagnosis. Liver function tests with the markedly elevated serum bilirubin, alkaline phosphatase, and cholesterol suggested an obstructive lesion. One entity best fitting this contradictory evidence is "cholangiolitic hepatitis," which is itself controversial and unacceptable as a diagnostic term to many pathologists.

Direct inspection of the extrahepatic biliary tract, as here, fails to show obstruction in this situation. Intrahepatic biliary stasis does seem to exist, but the assumption that real intrahepatic obstruction is present is questionable.

Most unusual in this case was the peculiar involvement of the nails and terminal phalanges; seemingly abnormal cholesterol deposition is here the explanation.

Pathological Anatomical Diagnosis

Primary

Cholangiolitic hepatitis.

Secondary hypercholesterolemia.

Secondary xanthomatosis with generalized ulceration and infection with Proteus, enterococci, staphylococci.

Lipoidosis of aorta, slight.

Bilirubinemia with cholemic neplosis, slight.

Altered blood in stomach and intestine.

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Answers to Tax Quiz

1. (b) On leased property, you normally spread the cost of improvements over the shorter period—the life of the improvement or the term of the lease. Since your lease expires in four years and presumably the furnishings will have a longer life than that, you should be able to claim a $250 deduction on your federal tax return for this year and the next three years.

2. (c) The money you received from the company must be reported as income, but you may deduct the cost of moving your entire family. If the amount the company gives you exceeds your expenses, the excess is taxable. Conversely, however, if your expenses were more than the amount received, the difference is not deductible.

3. (a) and (b) are both correct. All taxpayers are entitled to a $50 dividend exemption. A husband and wife can combine their exemptions and receive $100 in dividends tax free, providing the stock is jointly owned. The filing of a joint return will not qualify them for this double exemption if the stock is held in only one of their names.

4. (c) You cannot deduct contributions to an organization which spends a substantial part of its time lobbying or distributing political propaganda.

5. (a) You gained a son-in-law but lost a $600 dependency exemption for 1957 when your daughter married in November. All is not lost, however. If you provided more than one-half of your daughter's support during the year, you may claim her medical expenses as a deduction on your return.

6. (c) Watching television can be most relaxing and it might even help you to forget your poker losses—which is the thing to do because net gambling losses are definitely not deductible. Net gambling gains are taxable as income; if you won money in a football pool or other sources, you may use your poker losses to offset these gains.

7. (b) The IRS has ruled that "if the repairs do nothing more than restore the property to its condition immediately before the casualty and do not add to (its) value, utility or useful life, such repair costs may be used as a measure of the value of the destroyed portion." Where you were going at the time of the accident does not affect the deductibility of car damages.

8. (b) Commutation fees are not a deductible business expense. The cost of going to and returning from work, whether it be by bus, cab, train or plane, is not deductible since it is a personal expense. On the other hand, a and c are deductible.

9. Damage to your shrubbery caused by children, dogs or errant lawnmowers is not deductible. If your home or lawn is damaged by fire, storm or flood the loss not covered by insurance may be deducted. When large amounts are involved it is wise to have an expert appraisal made immediately after the casualty.

10. (b) Since your son works for *you,* you are not supposed to pay social security tax on his wages, nor is he required to make contributions. If your business is incorporated, however, the corporation must pay social security tax on his salary.

11. (a) Your deduction for a charitable contribution is the value of the gift at the time it is made. You are not considered to have realized a taxable gain or deductible loss when you give property away. You may claim a deduction for the entire $1,500 so long as this amount does not exceed 20 per cent (30 per cent in some cases) of your adjusted gross income.

12. (b) The roof is considered an improvement, not an ordinary repair. The cost of replacing the roof is deductible as depreciation spread over its estimated useful life.

13. (c) In the absence of fraud or substantial understatement of income, the Government has three years from the due date of your 1957 return to check your return and bill you for additional tax. Since the due date of most individual returns is April 15 and for investigation purposes all returns are treated as though filed on the due date, you should be sure to save all check stubs and receipted bills to prove your declared deductions until April 15, 1961.

14. (c) The fee which a CPA charges you to prepare a tax return or defend the accuracy of your return before the Treasury Department is deductible in full if you itemize deductions.

The Frozen Shoulder

Study of Its Anatomy, Symptomatology, Etiology, Pathogenesis, and Treatment

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The clinical entity today known as the "frozen shoulder" is commonly seen in general practice. However, in general, it is very poorly understood. The purpose of this paper is to present a brief but representative survey of the literature of some observations on the anatomy, symptomatology, etiology and pathogenesis, and treatment of this condition.

Anatomy

To help understand the clinical entity of the frozen shoulder, one must first become acquainted with the anatomical relationships of the various parts of the shoulder. The shoulder is a synovial joint with embryonal origin from mesenchymal tissue, organized actually as four joints working in harmony with ball and socket type action to give multiaxial function and smooth scapulohumeral rhythm with a range of motion seen in no other joint in the body.[16,20] The blood supply is from the anterior and posterior humeral circumflex and suprascapular arteries. Innervation is from the circumflex, suprascapular, and subscapular nerves.[16]

The joint is surrounded by a loose fibrous capsule into which tendons of the short rotator cuff muscles, the supraspinatus, infraspinatus, subscapularis, and teres minor, are intimately joined to form a musculotendinous cuff, which carries a large part of the weight of the arm. The muscles of elevation, because of the anatomy of shoulder position, do not have good leverage, so abduction is a comparatively weak function. The supraspinatus must be intact for deltoid function, and, similarly, the deltoid for scapular function.

The external surface of the joint, capsule, and musculotendinous cuff is covered by a bursa or gliding surface, the subdeltoid (subacromial) bursa.[18] There are eight bursa of the shoulder. The two most important clinically are the subdeltoid, which does not communicate with the joint space but lies just above the supraspinatus tendon, and the subscapular bursa, which is between the subscapular tendon and the articular capsule and does communicate with the joint capsule.[16] Hitchcock[19] emphasizes that contrary to popular opinion the tendon of the long head of the biceps which passes into the capsular ligament is fixed and the humerus moves on the tendon in a synovial pouch, especially in forward flexion and abduction.

Historical

Duplay in 1896 was the first to recognize and describe pathological disorders of the extra-articular structures as possible factors responsible for stiff and painful shoulders, and he called this entity "scapulohumeral periarthritis."[11] In 1934, Codman,[7] who had devoted much of his life to the study of shoulder lesions, found this entity "difficult to define, difficult to treat, and difficult to explain from the point of view of pathology." He termed it an adherent subacromial bursitis.

The term "frozen shoulder" crept into the literature in the early 1930's.[17] At various times and by various investigators, the entity has been called peritendinitis, periarticular calcifications,[6] para-articular calcification, subdeltoid or subacromial calcification, para-arthritis, Duplay's disease, calcic or calcific bursitis, soft-tissue rheumatism,[1] neuritis, fibrositis, capsulitis, adhesive capsulitis,[28] tendinitis, subacromial bursitis, supraspinatus tendinitis, and stiff painful shoulder. More recently Quigley[32] has coined the term "checkrein shoulder."

Etiology and Incidence

From the literature on the subject it is early evident that at the present date actually little is known of the etiology, pathology, or pathogenesis of this entity. Various agents and factors have been incriminated in the etiology of the frozen shoulder, and attention has been focused separately or conjointly on the following main factors: (1) a primary tendinitis, usually involving the long head of the biceps or the supraspinatus, with or without an associated subdeltoid bursitis; (2) calcifying tendinitis with calcareous deposits and a subdeltoid bursitis; (3) periarticular adhesions, and (4) adhesive capsulitis.[16] Codman[7] and others feel that anything which may cause spasm of the short rotator muscles or adhesions about the joint or bursa is covered by the term "frozen shoulder." Toxic, metabolic, and infectious factors have been im-

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Degner is now serving his internship at Lackland Air Force Base, San Antonio.

plicated in reports; however, much further investigation along this line is desirable. The old masters believed most cases of frozen shoulder were due to a type of tuberculosis of the joint, but they did recognize a special type that never suppurated, called it "caries sicca" and gave these patients an excellent prognosis.[17]

The syndrome of the frozen shoulder is usually found in people from 40 to 60 years of age with a peak incidence between the ages of 45 and 55. Stiff or frozen shoulder is becoming an increasingly frequent complaint, presumably because a greater percentage of the population is older, and Kitay[21] states that five per cent of all persons over the age of 40 years complain of a painful shoulder with limitation of motion at some time. Various authors have stated the sex incidence to be slightly greater in females, especially in the menopausal age, ranging from 50 per cent to 68 per cent.[11] Fletcher[16] stated the right shoulder is involved in two thirds of cases and is most often unilateral.[15]

Pathology

DePalma,[10] who operated on the shoulders of 42 patients, found a diffuse inflammatory process of varying severity involving all soft tissue components of the scapulohumeral joint, not restricted to a specific region of the joint. However, the biceps tendon was involved in all instances, whether primarily or secondarily, and this led him to believe that bicepital tenosynovitis is a causative factor in 65 per cent of cases.

Hitchcock[19] found that faulty anatomical mechanisms were responsible in a few cases such as a supratubercular ridge or a flat bicepital groove, which could initiate inflammatory process in and around the long head of the biceps tendon and eventually result in a painful and frozen shoulder.

Turek[41] in a survey study of the pathology of this condition found that a microscopic examination of the tissue of the cuff showed a diffuse chronic inflammatory process with degeneration of collagenous tissue, increased vascularity, cuffing of blood vessels, and mononuclear cell infiltration.

In 1949 DePalma[11] showed that the degenerative process starts as early as the second decade of life and is maximal in the sixth decade. The earliest noted lesions were an unevenness, fibrillation, and furrows in the tendons and cuff. Later this progressed to pitting erosion and marginal bone proliferation. The biceps tendon was especially noted to be involved in the fifth decade. The capsular degeneration leaves the long head of the biceps holding most of the weight of the shoulder, subjecting the biceps tendon to further minor traumatic incidents from which symptoms may evolve. Tearing, fraying, degeneration of collagen with fibrosis, and increased vascularity are progressive with age.[41] Alexander[1] verified the above pathological findings in his study.

Calcifications of the tendons of the musculotendinous cuff are noted in from 20 per cent[32] to 50 per cent[29] of cases. Newport[29] reported 20 per cent of his series due to subdeltoid bursitis and 10 per cent due to adhesive capsulitis.

Furlong[17] found tendon collagen infiltrated with fibrous tissue and chronic inflammatory cells with the blood vessels partially obliterated with small patches of avascular necrosis, more typical of ischemia than of infection generally. Although no serial histological data is available, it is presumed that this is a reversible process as most patients will recover spontaneously if left alone.

Young[42] states than in 53 per cent to 80 per cent of cases of painful and stiff shoulder, the bursa is involved in some manner, be it primarily or secondarily. Quigley[32] recently quoted "out of several hundred operations," "no torn rotator cuffs associated with stiff shoulder and no stiff shoulders associated with a torn rotator cuff" were found. However, many investigators include ruptured tendons of the rotator cuff muscles as a not infrequent cause of a frozen shoulder. Pathologically, a sterile inflammation usually about calcific deposits in the rotator cuff was found. Osteoporosis was noted in late cases.

Quigley[32] suggests the name "checkrein shoulder" because of the pathology he found of adhesive capsulitis involving principally the "anterior and inferior portions of the capsule" where gravitational effects caused the synovial fluids to be involved in adhesions acting as a checkrein to active and passive motion of the arm, especially abduction.

Pathogenesis and Symptomatology

The natural history of the frozen or stiff shoulder indicates that the syndrome is essentially self-limited and may have a clinical course lasting from a period of several months up to two to three years.[26] In following the clinical course of patients with stiff and painful shoulder, one finds in general that the four major factors involved in the pathogenesis of this condition are (1) age, (2) recurrent minor trauma, (3) pain, and (4) muscle spasm or immobilization of the shoulder joint with resultant muscular inactivity.

A typical case would be for a 50-year-old female typist going through her menopause to notice the gradual onset of a dull, toothache-like pain at the cap of the humerus or the insertion of the deltoid. The patient may blame her "rheumatism" on a slight cold, sitting in a draft, or some minor trauma, or she may have no explanation for it at all. Pain is more marked with movement, especially movements utilizing abduction, and may be noticed while putting on

a coat, combing hair, or reaching for objects above the height of one's head. The symptoms are usually progressive, but several months may elapse before the patient will report to a physician for relief. The onset may be acute, in which case there may be a history of slight strain or sprain or overuse. Pain is usually worse toward the end of a working day[7,21] or at night and may actually prevent sleep.

Drugs, including narcotics, may become ineffective in relieving pain, which may at times be unbearable, constant, and disabling[18] and may break even a strong man[7] or drive one to suicide. The patient is usually unable to lie on the affected side. The pain may radiate down the arm or into the neck and reflexly create spasm of the rotator cuff muscles, pulling in the head of the humerus high into the glenoid and produce immobilization which in turn tends to relieve the pain. Typically, the patient's extremity will not function in the elevated, externally rotated, and abducted positions, and the patient may involuntarily lose her grasp on objects when lifted into certain positions.

With pain and muscle spasm, voluntary movements become difficult to make. In approximately one week to one month or so, motion of the shoulder becomes definitely limited, especially external rotation[34] and abduction. Stoddard[40] states that immobilization for as short a time as two to seven days may result in a frozen shoulder with only scapulothoracic motion remaining. Varying degrees of limitation of motion exist from slight limitation of active motion in one axis to actual fixation of the shoulder and limitation of active and passive motion in abduction, flexion, and external rotation.

Clinical weakness of shoulder muscles may be demonstrable in three to four days if pain is present. Lack of movement allows adhesions to form in the bursa and shoulder joint from decreased normal circulation and synovial fluid production.[26] Farkas[14] and DePalma[10] believe that all soft tissue adjacent to and comprising the musculotendinous cuff becomes saturated with a serofibrinous exudate, and it is primarily the muscle arrest which induces venous congestion and edema, which eventually results in capsular, synovial, fascial, intermuscular, and intramuscular adhesions.

Coventry[9] believes a "periarthritis personality" plays an important role in pathogenesis in many cases. This type of person is hyperemotional with hyperactive vasomotor responses, is usually under physical and mental strain, is often tired out, restless, unstable, refuses to take the initiative, and often has a secondary anemia with a moderately accelerated erythrocyte sedimentation rate. DePalma[10] firmly believed the origin of pain was primarily the result of a tenosynovitis of the biceps tendon in approximately 65 per cent of cases. Turek[41] theorized that the pathogenesis lay with the basic mesodermal tissues degenerating with age and repeated minor trauma with a resultant decrease in elasticity and compression of soft tissue between the head of the humerus and the acromium process, which occurred especially with abduction and especially over the greater tuberosity. Edema, congestion, and fibrosis establish a cycle leading to fixation of the joint.

Factors favoring muscular inactivity are (1) involuntary splinting and immobilization which occurs secondarily to pain, and (2) voluntary immobilization sometimes instituted by a physician in treatment of some disease such as coronary heart disease, chronic lung disease, fractures of the arm, and even the painful shoulder, to name a few.

The scope of this paper is too limited to expound on conditions which may cause pain in the shoulder, and a number of conditions have been mentioned earlier in the report, but a few of the more common causes of a painful shoulder which may ultimately result in a frozen shoulder are: biceptial tenosynovitis, calcareous tendinitis (especially supraspinatus) with or without a secondary bursitis, sprain, contusions, fractures, and occasionally ruptures of the rotator cuff. Calcification in tissue does not always result in symptoms, as evidenced by Bosworth[6] in his study of more than 12,000 supposedly normal shoulders and finding 2.7 per cent with x-ray evidence of calcifications.

Once the patient voluntarily or involuntarily immobilizes the shoulder, the rapidity of the process increases.[17] Usually, at about six weeks, the pain starts to decrease and motion progressively begins to increase until normal mobility in all ranges is usually regained by six months. However, the whole cycle may take from 18 to 36 months. Full range of motion is recovered in 60 per cent to 100 per cent of untreated cases or "adequately" treated patients without complications. Those patients who do not fully recover usually have only mild to moderate limitation in the extreme ranges where practical function is of no great benefit unless there is some complicating factor. Late findings after prolonged immobilization are a mild but definite disuse atrophy of the supraspinatus and infraspinatus muscles[34] and disuse atrophy of bone. Atrophy roughly parallels the duration of disuse.[32]

Repeated insults, movement, and trauma during the acute or subacute stage lead to more splinting and muscle contractions, which results in overloading of the internuncial pool so that reflex muscle splinting spreads to involve other muscles innervated by the same cord segment. Since this fifth, cervical segment supplies many muscles with many different functions, the shoulder and arm may be extensively affected.[35]

Most observers agree with Codman that acute or

chronic trauma or overuse leads to rupture of a few fibers of some tendon, usually the biceps or supraspinatus, and to formation of small hematomas which subsequently calcify.[42] Shoso[36] suggests that a condition occurs in the capsule similar to that in the myocardium: a decreased vascularity with decreased carbon dioxide tension and an increased alkalinity resulting in precipitation of calcium salts and necrosis.

Diagnosis

To make an accurate diagnosis of the various lesions which can cause painful and subsequent frozen shoulder, one should have some knowledge of orthopedics, neurology, cardiology, and of diseases of the abdomen, thorax, and blood vessels. In evaluating this syndrome it is necessary to obtain a detailed history, including occupation, trauma, and details about pain, a complete physical examination of all systems, and x-rays if indicated. Often the diagnosis is made by the patient as a bursitis and is accepted and treated as such by the physician.[4] Young[42] states that frozen shoulder is associated with bursitis in 53 to 80 per cent of the cases.

To list or give a classification of the many etiological factors which may result in frozen shoulder is not the purpose of this article. If one can determine whether the source of difficulty is neurogenic, myogenic, arthrogenic, or vascular in origin and whether trauma, infectious, or mechanical factors are involved, one can usually narrow the differential diagnosis. Thoracic inlet syndrome and referred pain from the heart or other viscera should be ruled out.[31] If calcifications are noted by x-ray, one must rule out hyperparathyroidism as a rare but possible cause. It is important to diagnose ruptures of the rotator cuff early as these lesions may result in permanent dysfunction if not repaired by surgery. Ruptures can usually be diagnosed by eliminating the factor of pain and observing to see if the patient has active motion remaining in the range of motion supplied by the suspected ruptured muscle or tendon. The differential diagnosis of the cause of frozen shoulder is often difficult but important to make for reasons of treatment and prognosis, and a plea is made for physicians to acquaint themselves with the condition so that a fairly standard classification of diagnosis and definitive treatment may evolve.

Observations on Treatment

Treatment of the frozen shoulder is symptomatic, for the most part, and empirical. If adequate and early treatment of acute and chronic painful shoulder is instituted, one may not have a resultant "frozen shoulder." Lack of an early or accurate diagnosis accounts for many poor results reported in the literature, and treatment is of questionable value in the first place since this is usually a self-limited condition. The goal of treatment is (1) primarily to do no harm,[17] (2) to relieve pain, and (3) to restore muscular function and range of motion. Medication and therapy are used to try to break up the cycle in the pathogenesis of the frozen shoulder.

It is important to begin treatment as early as possible in order to relieve pain and permit early mobilization to prevent further adhesions from forming. Rest is of benefit in the relief of pain, and the patient may do this unconsciously. Rest does not mean immobilization, and Regan[33] feels that passive stretching exercises in all ranges of motion are indicated, increasing the range past the pain threshold each time at intervals of 30 minutes during the day to prevent further adhesions and help break up old adhesions. Rose[35] recommends rest in a functional position without immobilization, utilizing a bib sling for the forearm with avoidance of the triangular sling. Berg[3] recommends neck traction and posture correction in many of his cases.

Boland and Rosenberg[4] recommend acetylsalicylic acid or butazolidine as analgesics in cases where pain can be controlled by these medications. Codeine and strong narcotics are occasionally utilized temporarily to control acute pain, although there is danger in use of narcotics in any chronic condition. Relief of tension is important in these patients and may aid in quicker recovery. Duncan[12] emphasizes the importance of encouragement of the patient in exercises as the patient, not the physician, must perform them for expected benefits.

ACTH is also utilized in treatment because of its anti-inflammatory nature, and its side effect of producing euphoria also aids in management. Sedatives and muscle relaxants[35] are of value to some patients. Injection of trigger points with 2 per cent procaine often provides immediate relief from pain.[34]

Heat is utilized in various forms for its effect of increasing the blood supply to the area. If an acute inflammatory process is present, heat is frequently aggravating by causing added congestion in the area, and an ice bag may be of some benefit in such a case. Heat may be applied in the form of x-ray, infra-red, ultra-violet, diathermy, massage, ultrasonic, or histamine ointment, and each has its proponents. However, so far as is known, each is best used in conjunction with other types of therapy. Vasodilator drugs have been tried without dramatic results.

Shoso[36] believes that x-ray irradiation is the most rapid, convenient, and satisfactory method of treatment, especially in subacute cases, and he had only about 4 per cent recurrence utilizing the dosage; 140KV, 15MA, 50CM-TSD, 150r in air every other day for a six-day total of 450r.

Ultrasonic diathermy was not used in this country

until 1946, but since then it has gained some acclaim in benefiting up to 80 per cent of patients. Lehman[22] indicates that this is a good method of heating deep tissues and is beneficial in conjunction with other methods of treatment. He takes great care to avoid overdosage and subsequent destructive phenomena.[23] Contraindications to its use are malignant metastatic growths in the axilla, inflammatory infiltrations of the lung, or coronary heart disease.[23] Massage and histamine ointment are easily utilized for home treatment.[35]

Needling and injecting trigger points or calcifications is an office procedure and often provides immediate and adequate results. Occasionally two large needles are placed into the capsule and irrigation with normal saline to wash out calcium laden fluids and deposits is utilized. Steroids were first used in the treatment of this condition in 1951. Orbach[30] had excellent results with local injection of hydrocortisone acetate into the joint space, and Steinberg[39] had dramatic results utilizing ACTH 50 USP units twice on the first day and once each on the second and third days.

Murnahan et al.[27] used hydrocortisone with hyaluronadase 1M, not injecting into the joint, and had remarkable results with increased range of motion and relief of pain. Crisp[8] used a similar combination and noted 94 per cent of his patients improved with 72 per cent completely recovered in 7 to 12 days time. Hollander[20] had 852 patients in whom he injected intra-articular hydrocortisone and noted successful relief of symptoms in 85 per cent. Permanent relief was noted in 20 per cent but 54 per cent had recurrences. Results similar to those above have been noted from the oral ingestion of steroids.

Restoration of function is attained by the use of (1) physical therapy, (2) manipulation in selected cases, and (3) surgery. Active and passive exercises are begun as soon as the patient is able after the relief of pain. Progressively graded exercises to tolerance and past the point of pain in all ranges of motion are essential in preventing further adhesions and helping to break down adhesions already present.

Manipulation is of questionable value and is not utilized as frequently as it has been in the past although many reports indicate that relief from pain is obtained with manipulation under pentothal anesthesia.[1,2,15,32,33] DePalma[10] reported tears and ruptures of the cuff and tendons following manipulation and, like many other investigators,[17,25] now does not use manipulation except in select cases. If adhesions are present, more than likely the patient also has osteoporosis, and the humerus may easily be fractured by manipulation. Brachial palsy has been reported following manipulation.[25]

Many physicians do not recommend surgery except in some late chronic cases. Surgery has been used in excision of calcium deposits,[6] transplanting the biceps muscle to corocoid process in late cases,[6] and excision of the acromium process to provide for free motion in abduction.[10] Turek[41] lists the indications for surgery in this condition as: (1) failure to respond to conservative treatment with regard to motion and pain, (2) too slow improvement, economically undesirable to the patient, (3) recurrence, and (4) suspected gross damage to the cuff, tendons, or bony structure. Various combinations of the above mentioned methods of treatment have been utilized by various authors.

Summary and Conclusions

The "frozen shoulder" as a clinical entity is discussed with a brief but fairly representative survey of the literature. The anatomy of the involved area is reviewed, and a brief history of the entity is given.

Attention has been focused separately or conjointly on the following factors etiologically: a primary tendinitis, a calcifying tendinitis with or without a secondary bursitis, periarticular adhesions, and adhesive capsulitis.

The condition is found usually in people from 40 to 60 years of age and is increasing in frequency, presumably because a greater percentage of the population is in the older age group.

Histological pathology varies but in general shows a diffuse chronic inflammatory process with degeneration of collagenous tissue, sometimes beginning as early as the second decade but maximal during middle age. Often calcifications are found in and around frayed tendons, and occasionally complete rupture of tendons is noted.

The syndrome is usually self-limited and the clinical course may last from several months to several years. A cycle involving recurrent minor trauma, pain, and muscle spasm or muscular inactivity is established in the pathogenesis of this condition, and limitation of motion may involve any or all axes with limitation of active and passive motion.

Diagnosis is often difficult and is not stressed in this report. However, a plea is made for physicians to acquaint themselves with the syndrome so that a fairly standard classification of diagnosis and thus a more definitive treatment may evolve.

Treatment depends somewhat on accurate diagnosis, but is, for the most part, symptomatic. The goal of treatment is primarily to do no harm and to instigate measures to interrupt the cycle of pain and muscle spasm and immobility important in the pathogenesis of the syndrome. Physical therapeutic measures utilizing rest, heat, and exercises often used in conjunction with medicines such as analgesics, including needling and injection of trigger points, antispasmodics,

and steroids have been beneficial. Indications for surgery are listed.

The statistical data presented vary widely, even though an attempt was made to choose fairly representative articles on the subject with large series of patients. It is clear that much investigation and analysis of cases are desirable in order to increase our present knowledge of the frozen shoulder syndrome.

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Chemotherapy in Tuberculosis

(Continued from Page 10)

anism with a resulting decrease in the manifestations of illness. In patients seriously ill with tuberculosis of long duration there is evidence of adrenocortical hypofunction. Steroid therapy used with concomitant antituberculosis chemotherapy often effects striking symptomatic improvement. Thus, without anticipating any change in the ultimate outcome, the use of steroids would appear to be justified, if only for its symptomatic effect, in patients hopelessly ill with advanced tuberculosis. In acute forms of tuberculosis associated with severe clinical illness, steroids may be helpful. This is especially true of miliary and meningeal tuberculosis. In the latter condition, prevention and relief of cerebrospinal fluid block has been attributed to steroids.

Close to 500 children under 14 years of age are fatally poisoned each year in the United States, according to a report from the Kansas State Board of Health.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Eleven years ago, in passing the National Employment Act of 1946, Congress provided for two organizations whose sole function is to promote maximum employment, maximum production, and maximum purchasing power. One is Congress' own Joint Economic Committee; the other, the President's Council of Economic Advisers.

The President's Council constantly studies all forces —social as well as financial—that affect employment and production, and before each January 20 makes its report to the President, who in turn utilizes that in drafting his annual economic report to Congress.

At the same time the Congressional Joint Economic Committee is making its own separate studies, holding hearings and preparing a background of information against which to judge the President's economic recommendations when they come before it. The Congressional committee, however, is wholly advisory; it does not itself draft legislation but makes public its annual report before each March.

Although this committee is denied legislating power, its influence often directs the course of legislation. For example, a strong, one-page report from this committee is credited with keeping Congress in session after start of the Korean war and thus preventing a scheduled decrease in taxes.

When it calls in witnesses, the Joint Committee attempts to obtain a broad cross-section of opinion—the liberal along with the conservative. For this reason, recent hearings under sponsorship of the Joint Committee attracted more than casual interest. They brought together conflicting general philosophies and controversial specific issues. In the health-welfare fields, the following were some of the views:

The question of hospitalization for the retired aged through the Social Security mechanism was debated pro and con by the panelists. Two views:

Prof. Wilbur Cohen, University of Michigan: The former Social Security official maintains that the system can stand the drain of hospitalization for the aged. It could be done for one half of 1 per cent of taxable income, he argued, and he would raise the latter to the first $6,600 of income instead of the present $4,200.

W. Glenn Campbell, American Enterprise Association: Congress should give the medical profession and the insurance industry a chance to work out this problem through traditional methods rather than institute a costly compulsory system with all its attendant damage to the effective practice of medicine.

Two other panelists expressed parallel views on the broader and philosophical aspects of health and welfare:

Secretary Folsom of HEW: The burdens of disease, disability, ignorance and insecurity cannot be escaped by under-investment in health, education and welfare. Such an under-investment would have a costly effect on private charities, budgets of governments, efficiency of industry and the purchasing power of consumers.

Professor Clarence D. Long, Johns Hopkins University: An expansion of social welfare programs will have a very great stimulating effect on the economy, provided we play down those programs that involve mere charity and emphasize those that help people help themselves.

On the day of the hearing on health, education and welfare, the panelists agreed that no cash programs in education were called for despite the scientific manpower shortages. Other comments on education:

Professor Paul J. Strayer, Princeton University: Either federal aid will be forthcoming on terms that can be acceptable to the states or we will suffer a general deterioration in the quality of education.

President Howard R. Bowen, Grinnell College: Federal aid should not be granted directly to colleges and universities but through intermediary non-profit corporations controlled by boards of trustees made up of distinguished citizens.

Notes

A possible indication of legislation in 1958 comes from a December tour of southern medical schools by members of the House Interstate and Foreign Commerce Committee's health subcommittee. Among other things, they were concerned with the schools' need for more laboratories and classrooms.

The Department of Health, Education, and Welfare has started a 12-year study on the activities of a group of 3,000 newly retired men and women.

Between July 1 and mid-December, almost half the population of the country had been taken ill with an upper respiratory condition, including Asian influenza.

In its first year of operation, Medicare spent $43 million, with $22 million going to civilian physicians and $21 million to civilian hospitals; administrative costs ran about 3 per cent. Some claims are still pending.

PHYSICIANS' ACTIVITIES

Dr. I. J. Waxse, Oswego, was speaker at the November meeting of the Woman's Auxiliary to the Labette County Society in Parsons. His subject was "Physical Fitness of American Youth."

Members of the staff of St. Rose Hospital, Great Bend, honored **Dr. Marion F. Russell** at a dinner on November 21 in recognition of his 50 years of practice. Also attending the dinner were Dr. Russell's two sons, **Dr. Homer Russell,** who practices in association with his father, and **Dr. M. F. Russell, Jr.,** of Rochester, Minnesota.

Dr. Salvatore A. Scimeca, formerly of Caney, is now practicing in Axtell.

A plaque in recognition of many years of service as fire department physician was presented to **Dr. Francis S. Carey,** Kansas City, recently. The inscription read "Dr. Francis S. Carey, in sincere appreciation of his many years of unselfish service to the firemen. From Firemen of KCK Local 64, I.A.F.F."

Dr. Murray C. Eddy, Hays, has been elected to the board of governors of the American College of Surgeons.

Plans to retire on January 1 were announced last month by **Dr. Herschel R. Turner,** Hope. The physician, who is 80 years old, has practiced in Hope for more than 40 years.

Dr. James Brown of Glasgow, Scotland, has joined the medical staff of the Southeast Kansas Tuberculosis Hospital in Chanute. Educated at Glasgow University, Dr. Brown has been working in the field of tuberculosis since 1954.

Dr. Paul H. Lorhan, of the University of Kansas Medical Center, has been asked to conduct a course on geriatric anesthesia at a meeting of the American Society of Anesthesiologists in New York, March 9.

Dr. Henry O. Marsh, Wichita, attended the annual meeting of the American Academy of Cerebral Palsy in New Orleans, November 24-27, and was elected to active membership in the organization.

The Reno County Board of Commissioners has employed **Dr. Jay Armstrong,** whose former address was the University of Kansas Medical Center, Kansas City, to serve as county physician with headquarters in Hutchinson.

Dr. Ernest C. Moser, Holton, spoke to the Rotary Club in his home city on November 22 on the present status of medical education.

The American College of Physicians announces that five Kansas physicians became fellows at its meeting in Philadelphia last month: **Dr. Max Scott Allen** and **Dr. Robert A. Jordan** of Kansas City, **Dr. Robert S. Wallerstein** of Topeka, **Dr. George Loren Norris** of Winfield, and **Dr. Russell M. Wilder, Jr.,** of Topeka. Named as associates in the college are: **Dr. James E. Crockett, Dr. Martin J. FitzPatrick,** and **Dr. Charles M. Poser** of Kansas City, **Dr. H. Alden Flanders** of Hays, **Dr. Bernard H. Hall** of Topeka, and **Dr. Thomas J. Coleman** of Wichita.

Dr. William Burger, who succeeds **Dr. Francis Carey** as physician to the fire department in Kansas City, was a guest of honor at the annual ball of the Firemen's Relief Association in Kansas City last month.

Dr. Charles B. Wheeler, Jr., has been appointed instructor in pathology and oncology at the University of Kansas School of Medicine. Dr. Wheeler, a graduate of the school in 1950, has had residencies in pathology at St. Luke's Hospital in Kansas City, Missouri, at the University of Maryland in Baltimore, and at the Veterans Administration Hospital in Wadsworth.

"Parotid Carcinoma—Aids in Solving a Dilemma in Management" was the subject of a paper presented by **Dr. David Robinson,** of the University of Kansas Medical Center, at a Pan-Pacific Congress of the American Society of Plastic and Reconstructive Surgery in Honolulu in November. He was assisted in preparing the paper by **Dr. James Boley, Dr. Creighton A. Hardin,** and **Dr. Herbert J. Forrest.**

Dr. Richard E. Bartman, acting clinical director of the Parsons State Hospital Training Center, was speaker at the November meeting of the Shawnee County Association for Retarded Children in Topeka. He discussed new concepts in care, treatment, and training of the mentally retarded.

"Treating Overweight Patients" is the subject of a paper written by **Dr. George L. Thorpe,** Wichita, and published in the November 16 issue of the *Journal of the American Medical Association.*

Dr. D. Cramer Reed, Wichita, presented an exhibit, "Infertility in the Male," at the mid-winter meeting of the American Medical Association in Philadelphia last month.

Plans to discontinue practice in Garden City were announced last month by **Dr. Arnold H. Greenhouse,** who has accepted a position on the faculty of the school of medicine at the University of Colorado.

Dr. Ray T. Parmley, Wichita, has been named chairman of the Membership Committee of the American Society of Anesthesiologists.

New on the staff of Topeka State Hospital is **Dr. Richard E. Bartman,** acting clinical director of the Parsons State Hospital during the past two years.

Dr. Alexander Robert Chalian, Topeka, and **Dr. Louis Barrick Wilson,** Kansas City, became fellows of the American Society of Anesthesiology at its recent meeting in Los Angeles.

Dr. Paul H. Nitschke, formerly in private practice in Prairie City, Oregon, has joined the staff of the Veterans Administration Hospital in Topeka, to specialize in urology. He is a diplomate of the American Board of Urology.

Since 1900 heart disease has become more than ever a disease of middle and old age, Health Information Foundation says. Today about 70 per cent of all deaths from this disease take place at ages 65 and over, and another 25 per cent between the ages of 45 and 64.

DEATH NOTICES

Walter Peter Stoltenberg, M.D.

Dr. W. P. Stoltenberg, 75, an honorary memmer of the Edwards County Society, died in the Edwards County Hospital on November 17 after a long illness. He received his medical degree from State University of Iowa College of Medicine in 1905 and began practice in New Liberty, Iowa. In 1908 he moved to Kinsley and remained in practice there until his retirement.

Francis Marion Shonkwiler, M.D.

A veteran Emporia physician, Dr. F. M. Shonkwiler, 83, died at Newman Memorial Hospital in Emporia on November 24. He had suffered a broken hip in a fall at his home the week before. A graduate of Louisville Medical College, Kentucky, in 1898, Dr. Shonkwiler had lived in Emporia for 30 years and had practiced in El Dorado before that time. He was an honorary member of the Lyon County Society.

Llewellyn Magellan Hinshaw, M.D.

A physician in Bennington since 1904, Dr. L. M. Hinshaw, 82, died at his home on November 28 after an illness of several months. He was an honorary member of the Saline County Society. After graduation from Kansas Medical College in 1905, Dr. Hinshaw began practice in Bennington, where he remained except for a short time spent in Solomon. He was guest of honor at a community celebration in 1952.

Spencer Norman Chaffee, M.D.

Dr. S. N. Chaffee, 85, who had practiced in Dickinson County more than 50 years, died on December 11 at his home in Solomon. Since his retirement in 1954, because of poor health, he had been an honorary member of the Dickinson County Medical Society. He was graduated from the University of Illinois College of Medicine in 1904 and began practice in Morganville. He moved to Talmage in 1905 and to Solomon in 1939. Among his survivors are two sons who are physicians, Dr. Dean Chaffee of Abilene and Dr. Lynn Chaffee of Boston.

COUNTY SOCIETIES

Dr. John F. Coyle, Coffeyville, was named president of the Montgomery County Medical Society for 1958 at a recent meeting of the group. Serving with him are: Dr. Albert E. Bair, Independence, vice-president; Dr. Kenneth R. Grigsby, Coffeyville, secretary, and Dr. Gerald C. Bates, Independence, treasurer.

Officers for the Dickinson County Society were elected at a meeting held at Hope on November 21: president, Dr. James O. Gilliland, Herington; vice-president, Dr. A. W. Butcher, Abilene; secretary-treasurer, Dr. George C. Steinberger, Abilene.

Members of the Shawnee County Society entertained their wives at a dinner dance at the Topeka County Club on December 4. Dr. W. M. Mills, who received his medical degree 50 years ago, was elected to membership in the 85-50 club.

Dr. Charles S. Davis, Galena, has been elected president of the Cherokee County Society. Other officers are: Dr. Paul H. Grubb, Galena, vice-president; Dr. Richard H. Claiborne, Baxter Springs, secretary-treasurer, and Dr. Gregg B. Athy, Columbus, delegate to the state meeting.

Dr. Eugene Schwartz, Dodge City, was guest speaker at the November meeting of the Iroquois Medical Society, held at the home of Dr. Jack E. Randle, Bucklin.

A meeting of the Cowley County Society was held at the Country Club in Arkansas City on November 21. Dr. Robert P. Holt, Oklahoma City, discussed treatment of deformities in children's feet.

Serving as 1958 officers of the Crawford County Society, as the result of an election held at the Hotel Besse, Pittsburg, in November, are: Dr. Clifford B. Newman, Pittsburg, president; Dr. Jack D. Walker, Girard, vice-president; Dr. Robert W. Friggeri, Girard, secretary-treasurer; Dr. Paul B. Leffler and Dr. Clarence M. Montee, both of Pittsburg, members of the censor committee. Dr. Clarence W. Erickson, Pittsburg, will serve as the group's Blue Shield representative.

"Recent Advances in the Treatment of Acquired Valvular Disease of the Heart" was the subject discussed by Dr. Dwight Harken, associate clinical professor of surgery, Harvard Medical School, before a meeting of the Sedgwick County Society in Wichita on December 3. At an afternoon clinic at St. Francis Hospital, the subject was "Traumatic Injuries of the Chest and Carcinoma of the Lung."

Members of the Labette County Society were guests of the Auxiliary at a dinner at the Parsonian, Parsons, on November 13. Dr. Rolland W. Urie was speaker at a separate meeting of physicians following the dinner and showed a medical film.

Dr. and Mrs. Orlin W. Longwood, Stafford, entertained members of the Stafford County Medical Society and its Auxiliary at a dinner meeting at their home in November.

Members of the Shawnee County Medical Society provided polio shots for $1.00 each at a clinic in Topeka on December 12. The program, widely acclaimed in Topeka newspapers as a public service project of the society, was called "Polio Dollar Day."

Dr. and Mrs. Clemens M. Rucker entertained members of the Nemaha County Society at a dinner in Sabetha last month, the occasion marking Dr. Rucker's 50th year in the practice of medicine. A film, "Grand Rounds No. 5," was shown by Mr. Del Saunders of the Upjohn Company.

Dr. Laurence S. Nelson, Jr., who completed his term of office as president of the Saline County Society last month, entertained the group at a cocktail party and dinner at the Warren Hotel in Salina on December 12. Dr. Frank T. Cultron is serving as president of the group for 1958.

A quarterly meeting of the Central Kansas Medical Society was held at the City Building in Russell on December 12. Dr. Wu Hao Tu and Dr. Robert Jordan, members of the faculty at the University of Kansas School of Medicine, presented a scientific program.

The annual Christmas Party for members of medical societies in an eight-county area was held at the

Neodesha Legion Hall on December 11. Wives of the physicians were also present for the dinner and program.

Dr. George A. Patton became president of the Atchison County Society as the result of an election held recently. Dr. Ira R. Morrison is vice-president and Dr. Wayne O. Wallace is secretary-treasurer. Dr. Wallace also serves as delegate to the state meeting with Dr. Charles H. Young as alternate.

BOOK REVIEWS

The Chronically Ill. By Joseph Fox, Ph.D. Published by Philosophical Library, Inc., New York. 229 pages. Price $3.95.

According to the jacket blurb Dr. Fox, whose advanced degrees are in social science, has devoted almost 20 years to the study of the aged and the chronically infirm. The publishers confidently state that the material "is of great interest to the busy physician, the social worker, the hospital administrator and to labor and management." It is possible that the social worker and others may find Dr. Fox's study of great value and interest, but the busy physician will probably not get any startling new facts or ideas from it because he daily faces the problems of chronic illness at a much less superficial level than Dr. Fox does.

It is also unlikely that this book, which "is a sociological approach to the problem" of chronic illness, "will evoke in others a kindred awareness of the problems and the goal that we should all strive for in our struggle to master and conquer the ravages of chronic disease." True, it does take a daring and straightforward stand in favor of viewing the chronically ill as human beings with psychological, social, and economic problems rather than as creatures to be herded into an asylum or put off in some back room. The radical nature of this idea might have surprised physicians in some past era, but one cannot imagine its being any more revolutionary today that would be a sermon against sin.

The book is printed on cheap paper and rather poorly bound. The bibliography will not be of much help to physicians, and the glossary is aimed at a mental age of approximately 12 years.—*J.D.R.*

Ear, Nose, and Throat Dysfunctions Due to Deficiencies and Imbalances. By Sam E. Roberts, M.D. Published by Charles C Thomas, Springfield, Illinois. 323 pages; 57 figures. Price $8.50.

Throughout his book, Dr. Roberts accentuates the importance of cellular nutrition and metabolism in diseases and dysfunctions rather than the virulence and toxicity of the micro-organism.

The foreword is by Dr. Morris Fishbein, who states: "Dr. Roberts has wisely recognized that conditions affecting the nose, throat, the sinuses, the nervous system—in fact, any portion of the human body—are in a sense not localized but constitutional disorders . . . and attack on such should not be limited to surgical correction of anatomical errors nor to eliminations of infectious or toxic agents. The attack must be total. His extensive experience using total approach to the control of dysfunctions of the sinuses, Meniere's disease, headaches, allergies, tinnitus, exhaustion, fatigue, and many other conditions has apparently yielded excellent results."

The sinus mucosa and the acoustic labyrinth are two extremely sensitive barometers which register systemic metabolic disorders occurring elsewhere in the body.

The author is certain that deficiencies, and especially imbalances, are the principal causes of the diseases, dysfunctions, and syndromes reported in his book and that they are often precursors of many chronic degenerative diseases. These deficiencies and imbalances are nutritional, electrolytic, hormonal, acid-base, and insulin-sugar. To correct one deficiency or imbalance and disregard others (since they are always multiple), would mean therapeutic inadequacy and lead to disappointing clinical results. Adequate nutrition heads the list of basic therapeutic procedures.

Chapter headings include: The Therapeutic Test, Philosophic and General Discussion, Clinical Observation, Notes on Specific Minerals, Carrel-Lindberg Heart, Antimetabolites, Gonadal Imbalances, Vascular Headaches (an excellent chapter), Insulin-sugar Imbalance, Meniere's Disease, Acoustic Nerve Dysfunction, Hilger's Syndrome, Allergy, Sinus Dysfunction, and Basic Therapy (well outlined).

Among the figures presented are: questionnaires on personal history, diet history, and gynecic history; general instructions for the allergic patient; list of food selection for adequate nutrition with general instructions; table of hidden sugars in foods; glucose tolerance charts and audiograms illustrating therapeutic responses. Case histories and chapter summaries are of interest and value.

The presentation is informal, easy to read, and easy to understand. It already has changed our own family's dietary regime.—*R.M.-S.*

The Story of Peptic Ulcer. By Richard D. Tonkin, M.D., F.R.C.P. Westminster Hospital, London. Published by W. B. Saunders Company, Philadelphia and London. 71 pages. Price $2.25.

Doctor Tonkin has incorporated into this small book a wealth of scientific material and medical advice written in a delightful manner. It is pleasurable reading for members of the profession and laymen alike and can be read in its entirety in 30 minutes. It would be an excellent guide to the multitude who suffer from peptic ulcer and I believe would give them a better understanding of their disease than would several hours of advice in the consultation room.—*C.C.G.*

Fads and Fallacies in the Name of Science. By Martin Gardner. Published by Dover Publications, Inc., New York City. Paperbound. 363 pages. Price $1.50.

The subtitle tells the story of the book—"The Curious Theories of Modern Pseudoscientists and the Strange, Amusing and Alarming Cults that Surround Them—A Study in Human Gullibility."

This book is not fiction. It is not written in a popular style. It is not intended to amuse. As we read the book we received the impression that this serious science writer is trying to compile a synopsis of the life and theory of each major cultist. Some receive a scant paragraph while others are dealt with at length.

Perhaps the major problem encountered by this reviewer was that of extreme brevity. The author tried to describe a complex philosophy in three sentences and that cannot be done. The alternative of course would be to expand these 383 pages to many volumes.

The book, then, is almost a dictionary, more accurately, perhaps, an encyclopedia of unscientific theories to which people have subscribed. Only a portion deals with health. Many treat the end of the world, biblical prophecy, racial superiority, life on other planets, sex, divining rods, etc.

Among them are some that are familiar but also to be found here are weird, bizarre beliefs this reviewer had never heard of. Included too are all the medical cults, food faddists, orgonomy, and dianetics. Named are such figures as Brinkley, Koch, W. H. Bates, Macfadden, and many others.

The author is not beating the drum for medicine. He is not fighting for or against any man or any theory. He is pleading for the scientific approach and in this effort exposes those who have ignored that discipline. Some cultists are out-and-out frauds, others sincere but misguided, and still others are in the mad genius class. All are reviewed.

The book is an excellent compilation of data a physician should have at his disposal. The book is recommended, perhaps not to be read continuously as a novel but to be sampled as an encyclopedia for information.—*O.E.E.*

ANNOUNCEMENTS

First Oklahoma colloquy on advances in medicine (fluid, electrolyte, and nutritional balance), auditorium, University of Oklahoma School of Medicine, Oklahoma City, February 6-8. Open to all physicians. Registration fee $25.

Board of Missions of Methodist Church needs 20 doctors in its mission fields in 10 overseas countries. Board now has 42 physicians in Asia, Africa, North and South America with 43 others in training. Information available at 150 Fifth Avenue, New York 11, New York.

May 1 deadline for essays in competition sponsored by Mississippi Valley Medical Society, winner to receive $100, a gold medal, and a certificate. Any subject of medical or surgical interest acceptable. Information available from Harold Swanberg, M.D., 209 W.C.U. Building, Quincy, Illinois.

Eleventh annual postgraduate course on diseases of the chest, sponsored by American College of Chest Physicians, Warwick Hotel, Philadelphia, March 3-7. Tuition $75. Write Director, 112 East Chestnut Street, Chicago 11, Illinois.

New York University-Bellevue Medical Center courses in February: congenital heart disease, February 3-5; auscultation of the heart, February 24-26; cardiac roentgenology, February 27-28; dermatology and syphilology, February 17-21; orthopedics in general practice, February 19-21; review in ophthalmology, February 20-22; review in pediatrics, February 17-22; otolaryngology, February 17-21. Tuitions vary. Write Association Dean, 550 First Avenue, New York 16, New York.

Fifty-seventh annual meeting, Medical Library Association, Hotel Kahler, Rochester, Minnesota, June 2-6. Pre-convention activity includes refresher courses. Information available from Mr. Thomas E. Keys, Librarian, Mayo Clinic.

Twelfth annual symposium on fundamental cancer research, University of Texas M.D. Anderson Hospital and Tumor Institute, Houston, March 6-8.

Fifth congress, International Society of Internal Medicine, Philadelphia, April 23-26. First held in U. S. Information from Secretary-General, Dr. Edward R. Loveland, 4200 Pine, Philadelphia 4, Pennsylvania.

Fifteen-minute film, "Professional Education," available from National Foundation for Infantile Paralysis, 301 East 42nd Street, New York 17, New York. Stresses need for cooperation between professional and community groups.

Twenty-sixth annual alumni postgraduate medical convention, College of Medical Evangelists, Hotel Biltmore, Los Angeles, February 25-28, preceded by two-day series of refresher courses at the college. Write for program, 1720 Brooklyn Avenue, Los Angeles.

New Code Announced

A new revised Code of Ethics for hospitals and hospital administrators has been released by the American Hospital Association and the American College of Hospital Administrators. It supplants a code produced and adopted in 1939 by the two organizations.

The revision, prepared by a joint committee representing both groups, consists of a ten-point statement on hospital ethics and a ten-point statement of principles of conduct for hospital administrators.

According to the introduction to the Statement of Administrative Principles, "The ethical and administrative principles . . . are established to promote the public welfare and to guide governing bodies, administrators, physicians, nurses and all who work in hospitals."

"By following these principles," the introduction states, "the hospital will be better able to perform its functions in a manner that will merit and receive the endorsement and support of the community it serves."

Hospital Ethics

1. Recognizing that the care of the sick is their first responsibility and a sacred trust, hospitals must at all times strive to provide the best possible care and treatment to all in need of hospitalization.

2. Hospitals, cognizant of their unique role of safeguarding the nation's health, should seek through compassionate and scientific care and health education to extend life, alleviate suffering, and improve the general health of the communities they serve.

3. Hospitals should maintain and promote harmonious relationships within the organization to insure the proper environment for the considerate and successful care and treatment of patients.

4. Hospitals should appreciate and respect individual religious practices and customs of the patient.

5. Hospitals, to the extent possible, should conduct educational projects, stimulate research, and encourage preventive health practices in the community.

6. Hospitals should cooperate with other hospitals, health and welfare agencies, governmental and private, and other recognized organizations interested in promoting the health of the nation.

7. Hospitals in reporting their work to the public should give a factual and objective interpretation of accomplishments and objectives without disparaging the work of other hospitals or related organizations.

8. Hospitals should actively support and encourage every effective method which will ease the financial burdens of illness.

9. Hospitals should be fair, honest, and impartial in all their business relationships.

10. Hospitals should be progressive in politics, personnel practices, and efforts to maintain up-to-date equipment, methods, and standards of performance.

For Hospital Administrators

The hospital administrator's life is dedicated to the highest possible level of performance in the competent and humane hospital care of the sick, in health education in all its many phases, and in research conducted in the interest of hospitals and their patients. In pursuing these objectives the hospital administrator should be guided by the following principles:

1. He will not use his position or influence for selfish personal advantage or gain and will not disparage the work of his colleagues.

2. As official representative of the hospital's governing body and often the hospital's spokesman in the community, his conduct will at all times be dignified and exemplary. His professional performance will be objective and fair, with the patient's best interest as the ultimate consideration.

3. In his relationships with personnel and staff he will be impartial, tolerant, fair, and interested in all reasonable means of promoting personnel morale and welfare, consistent with the hospital's best interests and ability to provide them.

4. The administrator will encourage, assist, and teach others the principles and practice of hospital administration to the end that future hospital administrators may be more adequately prepared.

5. The administrator will encourage and participate, to the extent possible, in a broad educational program to assure the health workers necessary to the hospital field.

6. He will contribute his interest, support, and leadership toward the general improvement of the

community, with especial emphasis on health education and related causes. In so doing he will attempt to avoid involving his hospital in partisan political issues.

7. In his relationships with the medical staff of the hospital he will support that which is constructive, sound, and in the interests of good hospital professional practice; he will resist and oppose that which is, in his judgment, harmful, destructive, or unwise.

8. The administrator will seek constantly to improve his professional knowledge and skill and will accept counsel and guidance, particularly in fields and subjects with which he is not entirely familiar.

9. Recognizing that his is a position of public trust, he will, within the limitations imposed by good judgment, legal considerations, and his hospital charter respect the rights, privileges, and beliefs of others regardless of race, color, or creed. He will keep confidential whatever he may learn respecting the private affairs or character of patients and their families, physicians, and others with whom he is associated in the hospital. When his administrative duties bring him into conflict with any segment of society or belief, he will deal with the situation with the greatest consideration, courtesy, and respect for the individual that is possible, without ridicule or animosity.

10. He will exemplify the Golden Rule in thinking, action, and conduct.

"March of Medicine" TV Program

The work of American physicians in remote regions of the world where native populations are largely dependent upon our doctors and medicine for their health and well-being is the television story to be aired coast-to-coast January 23. Entitled "MD International," the hour-long show will be presented at 8 p.m. CST over the full NBC-TV network in color and in black and white. This is part of a joint American Medical Association and Smith, Kline and French Laboratories project to inform the American public of people-to-people activities in the health profession for the promotion of better international understanding.

The telecast reports on doctors' activities in thoracic and general surgery, orthopedics, ophthalmology and general medicine in such far-flung areas as Korea, Hong Kong, Burma, Sarawak, Nepal, India, Lebanon, and Ethiopia. A special March of Medicine team traveled more than 34,000 miles to film these doctors in their unofficial roles as America's "medical diplomats."

Two world-wide figures—Charles Malik, foreign minister of Lebanon, and Emperor Haile Selassie of Ethiopia—will express their peoples' gratitude for the American medical work being carried on in their countries.

ABSTRACTS FROM CURRENT LITERATURE

Diffuse Otitis Externa and Its Effective Treatment. By G. W. Lawson, Postgraduate Medicine 22:501 (Nov.) 1957.

Following a comprehensive discussion of the differential diagnosis and etiologic relationships involved in diffuse otitis externa, the author presents details of an effective method of treatment. He advocates prompt measures to alleviate pain and to control infection, because, he states, "the commonness of pain as the presenting complaint indicates that infection is usually present at the time the majority of patients seek the physician's help." He also points out that: "Although bacteriologic diagnosis is theoretically desirable in every instance, the average patient who comes to a general practitioner because of earache will not readily accept the delay, inconvenience and extra cost."

The author states that in practice, treatment with an agent whose established use is confined to local application is both safe and satisfactory. His best results have been attained with a special liquid preparation which has a physiologic pH of 6.5, contains 3.5 mg. neomycin (as sulfate) and 50 mg. sodium propionate per cc. of alcoholic glycerin vehicle. The spectrum of neomycin includes gram-negative and gram-positive organisms, notably Pseudomonas bacilli and hemolytic staphylococci which are the bacteria found most commonly in ear infections. Propionic acid is particularly effective against the fungi which have been reported present in about 25 per cent of these cases.

As a routine measure against pain, the author prescribes aspirin and codeine, or, if necessary, a more potent analgesic. He also advocates local cold applications and avoids hot applications.

Of 713 patients treated successfully by the author's method, 347 required only one office treatment; 237 required two visits; 87, three, and 32, four treatments. The remainder required more than four, but their ear conditions had been complicated by perforated drums and other effects of previous chronic otitis media. However, following application of the medication their ears became free of discharge and improvement in chronic otitis media was noticed.

Program to Facilitate Research

A six-part program has been proposed by a committee of the National Society for Medical Research to relieve the increasingly serious shortage of cadavers threatening the quality of medical education in the United States.

"A majority of medical colleges report that they are unable to obtain enough bodies to teach student doctors efficiently," Dr. Oliver P. Jones, head of the anatomy department at University of Buffalo Medical School and chairman of the committee, said. "Some schools have been forced to drop such important courses as surgical anatomy."

The committee's program includes:

1. A survey of public opinion toward anatomical studies.

2. A series of conferences with religious leaders, public welfare administrators, undertakers, hospital superintendents, and other persons concerned with the disposition of bodies.

3. A program of education for persons in the health professions.

4. A general public educational program.

5. The drafting of modern laws making bodies available through bequest (in 39 states, a person's body is not his own to give after death).

6. Legal reference service, with standardized forms and procedures for bequeathing a body to a medical school.

In the opinion survey, a depth-interview study that will discover underlying sentiments has been proposed. Results of the study will provide a foundation for the other five elements of the program.

Dr. Jones explained that "Changing times, plus outmoded laws and ancient prejudices, mean increasing trouble for the anatomy departments of many of America's medical colleges."

The University of Buffalo alone operated its anatomy department last year with 23 cadavers short of adequate instruction. It was not able to give anatomical instruction to nurses and people in the public health fields. The department used 37 bodies to instruct 68 dental and 80 medical students. "This means four students to each body," Dr. Jones explained.

"If we have to assign a larger number of students to each cadaver, as we will if the shortage continues, students will not get proper experience in dissection. And they will be inadequately prepared when they subsequently have to operate on living patients."

More than 96 per cent of vehicles involved in fatal accidents on U. S. highways in 1956 were in apparently good condition.

Home to KUMC Dean

A spacious English-style home in the Mission Hills section of Kansas City, at 5800 Mission Drive, was presented to the University of Kansas last month by Mr. and Mrs. Kenneth A. Spencer for use as a residence for the dean of the School of Medicine. Mr. Spencer is president of the Spencer Chemical Company, and both he and Mrs. Spencer attended the University of Kansas.

A.M.E.F. State Chairmen to Meet

The American Medical Education Foundation's 1958 fund raising drive for the nation's medical schools will be officially launched January 25-26 at a meeting for state chairmen. This seventh annual conference will be held at the Drake Hotel, Chicago.

As a departure from the usual practice, a get-together dinner will be held Saturday evening, January 25, and Sunday sessions will be devoted entirely to a discussion of ideas for the further development of A.M.E.F. campaigns during the year. New work kits will be distributed to state chairmen along with samples of materials used by various state committees. A.M.E.F. will pay the expenses of one representative from each state, although any physicians interested in this project are welcome to attend.

In addition, A.M.E.F. reports that Indiana and South Carolina physicians have endorsed a dues increase to help support medical education. Other states which already have adopted a special dues program are Arizona, California, Idaho, Illinois, Nevada, New Jersey, and Utah.

Institutes on Nomenclature

Plans for sponsorship of two institutes on nomenclature have been announced by the American Medical Association as the result of the popularity of previous courses. The first institute in 1958 will be conducted March 31 to April 2 in Tulsa, and the second will be held in July, on dates to be announced, in Boston. The sessions are planned as a service to medical record librarians and others working with the Standard Nomenclature of Diseases and Operations in hospitals, clinics, or doctors' offices. Queries on the courses should be sent to A.M.A. headquarters.

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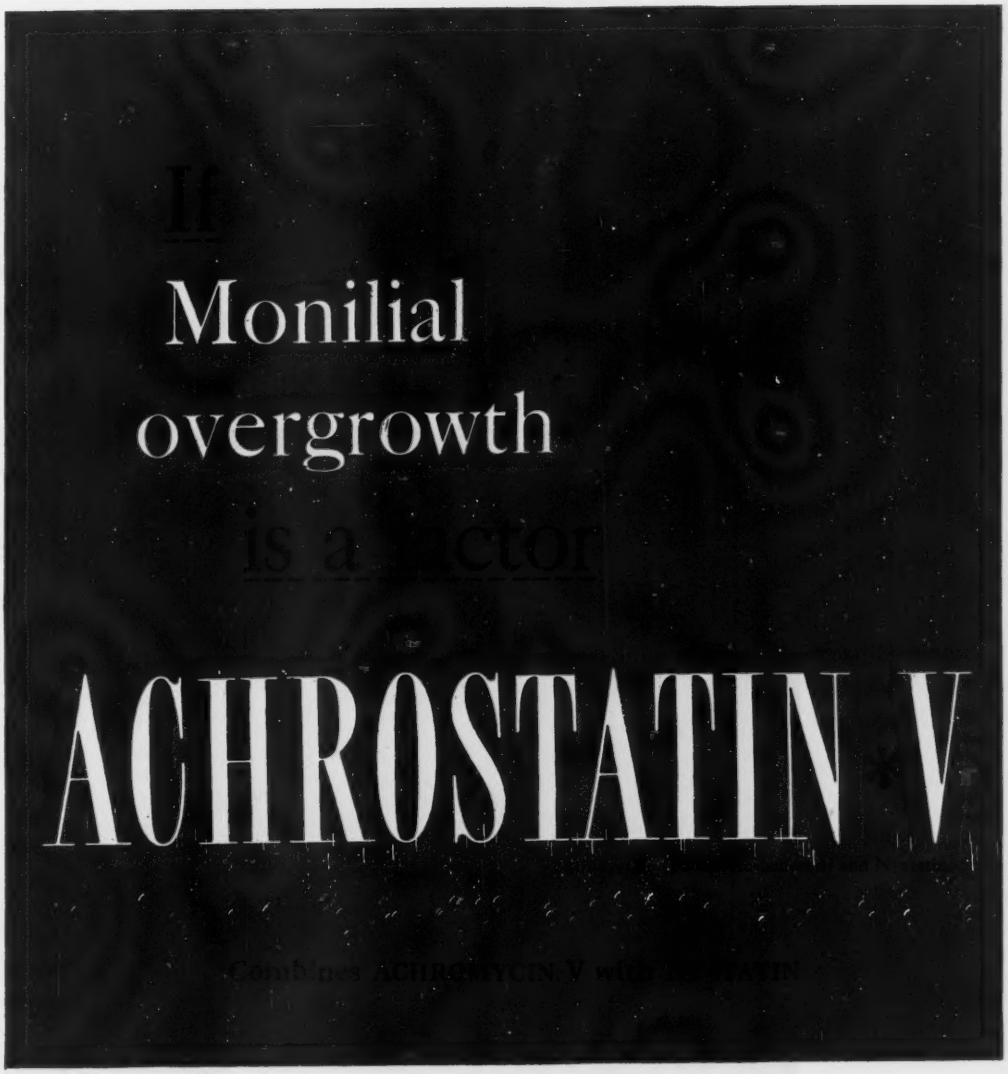

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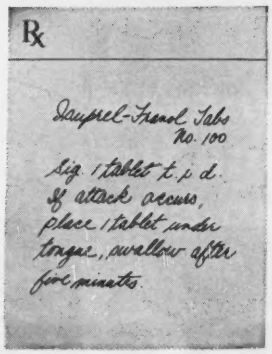
Isuprel-Franol tablets are "... effective in controlling over 80% of patients with mild to moderate attacks of asthma."[1]

1. Fromer, J. L., and DeRisio, V. J.: *Lahey Clin. Bull.* 10:45, Oct.-Dec., 1956.



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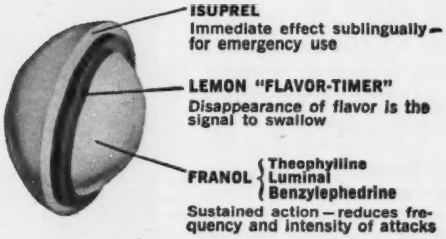

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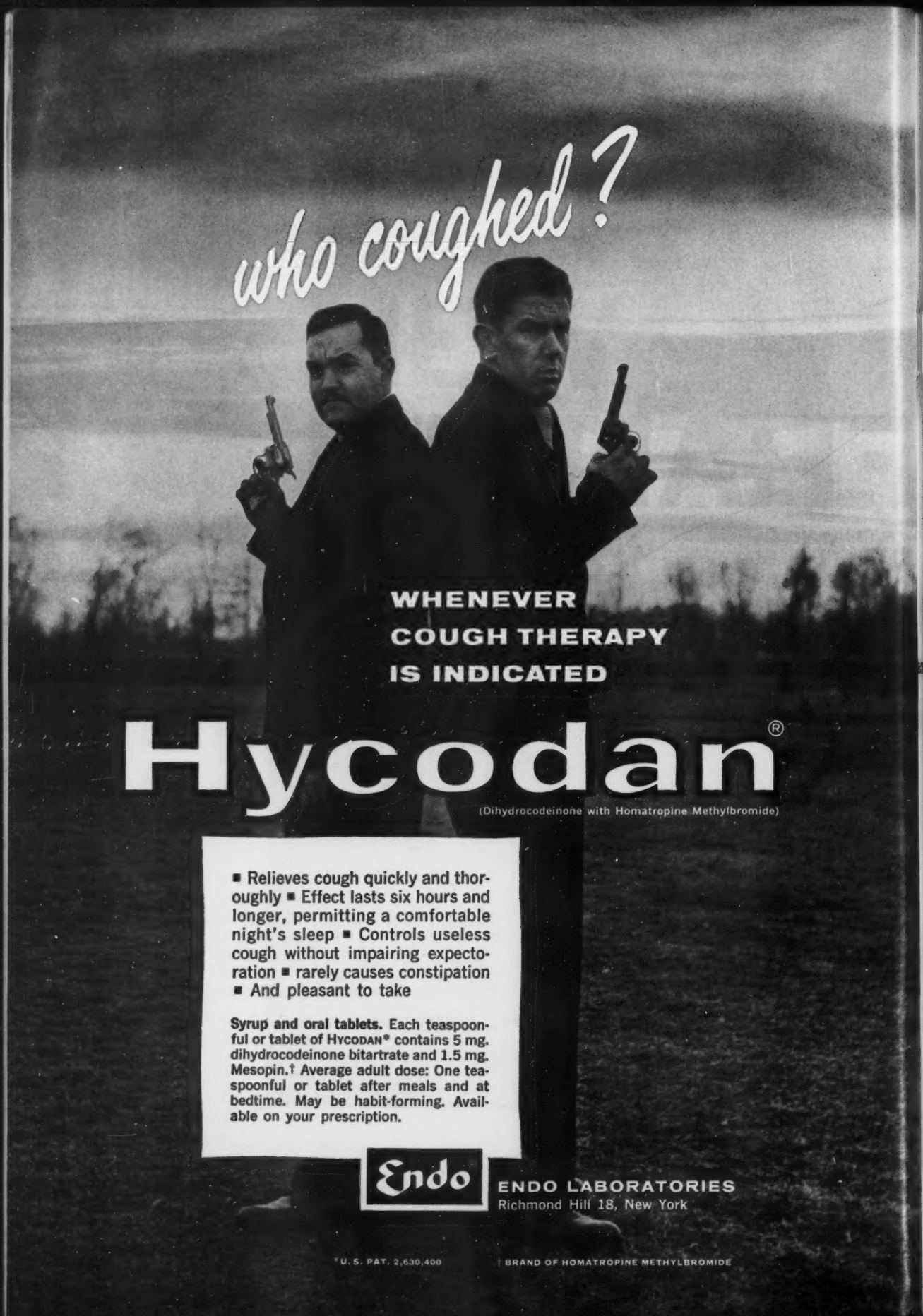
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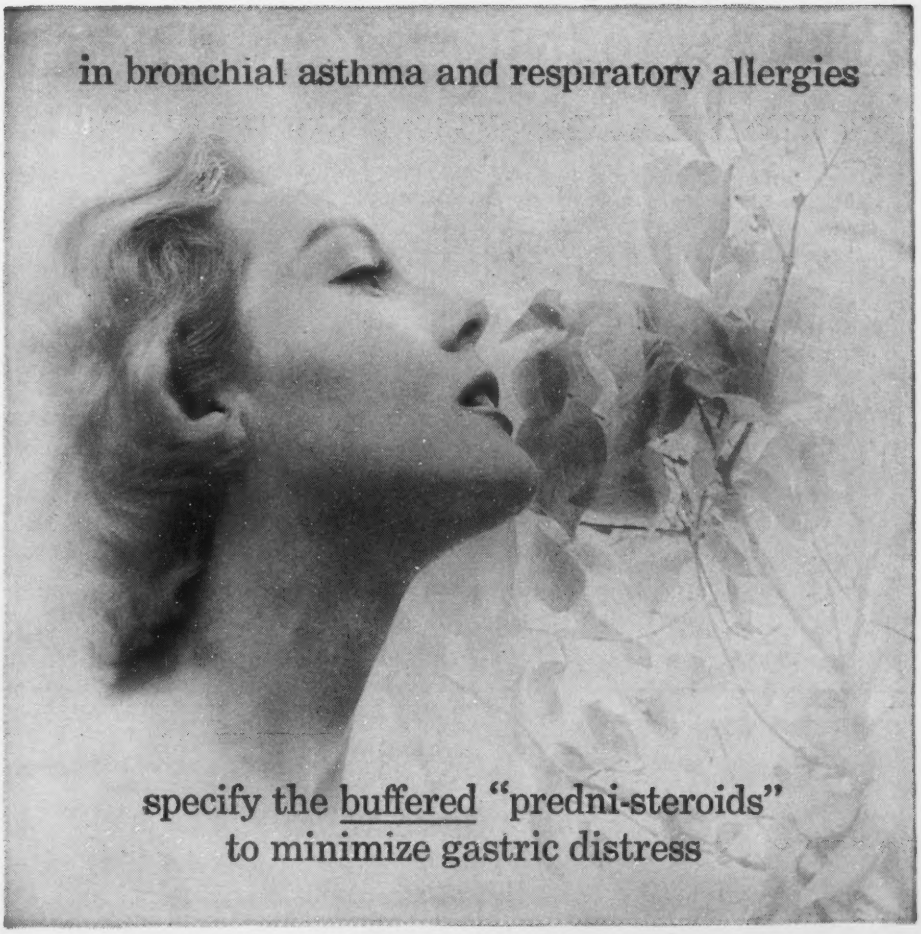

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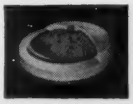

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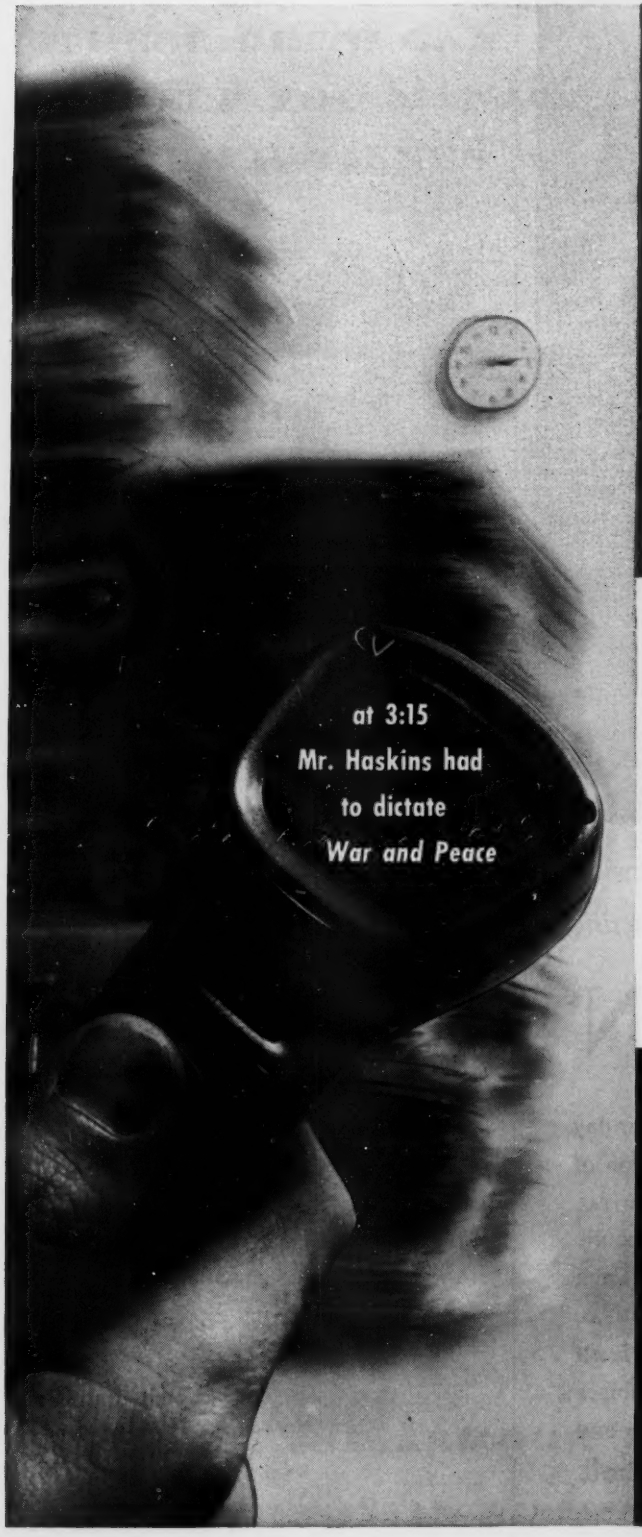

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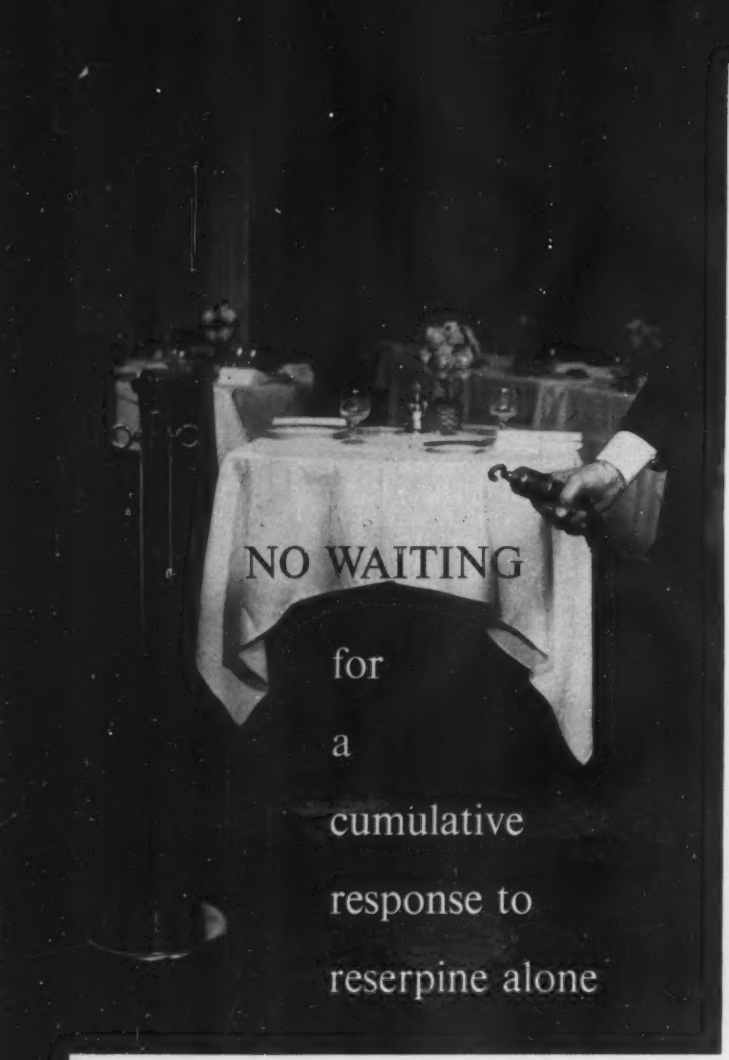
1. Comparative Effects of Various Rauwolfia Alkaloids in Hypertension, submitted for publication.



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NEW *fast-acting*

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Calmer days, more restful nights starting *first* day of treatment, through synergistic action of HARMONYL (Deserpidine, Abbott) and NEMBUTAL (Pentobarbital, Abbott). Lower therapeutic doses, lower incidence of side effects. Each HARMONYL-N Filmtab contains 30 mg. NEMBUTAL Calcium and 0.25 mg. HARMONYL. Each HARMONYL-N *Half-Strength* Filmtab combines 15 mg. NEMBUTAL Calcium and 0.1 mg. HARMONYL. Abbott

®Filmtab—Film-sealed tablets, Abbott; pat. applied for
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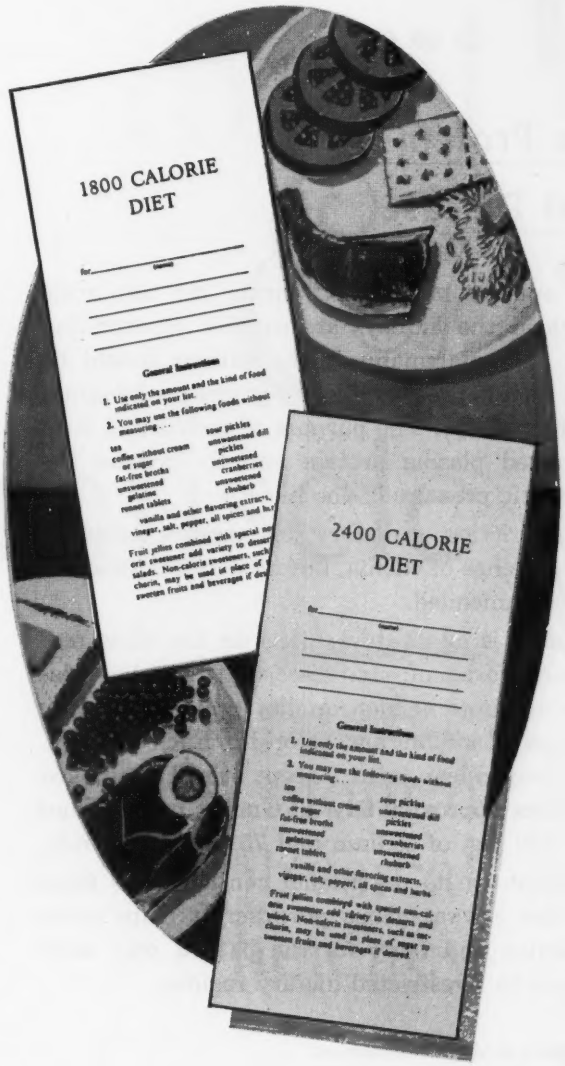

Upon your request, The Armour Laboratories will be pleased to send you a complimentary supply of 1800 and 2400 calorie diets . . . low in carbohydrate and high in unsaturated fats . . . intended for use in conjunction with ARCOFAC, the Armour preparation designed to lower elevated blood cholesterol.

Arcofac need be taken only once a day . . . in relatively small amounts . . . and allows the patient to eat a balanced, nutritious and palatable diet.

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Linoleic acid*. . . . 6.8 Gm.
Vitamin B_6. . . . . 0.6 mg.
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*derived from safflower oil which contains the highest concentration of unsaturated fatty acids of any commercially available vegetable oil.

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and the Protein Need in Renal Disease

Prevailing opinion holds that during the nephrotic state—provided the kidneys are capable of excreting nitrogen in a normal manner—the patient should be given a diet high in protein (1.5 to 2 grams per kilogram of body weight daily). The purpose of such a diet is to replace depleted plasma protein and to increase the colloidal osmotic pressure of the blood.

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Lean meat is admirably suited for the diets prescribed in most forms of renal disease. It supplies relatively large amounts of high quality protein and only small amounts of sodium and chloride. Each 100 Gm. of unsalted cooked lean meat (except brined or smoked types) provides approximately 30 Gm. of protein, and only about 100 mg. of sodium and 75 mg. of chloride.

In addition to its nutritional contributions meat fulfills another advantageous purpose: It helps make meals attractive and tasty for the patient who must rigidly adhere to a restricted dietary regimen.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

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relaxes both mind & muscle

without impairing mental or physical efficiency

well tolerated, relatively nontoxic / no blood dyscrasias, liver toxicity, Parkinson-like syndrome or nasal stuffiness / well suited for prolonged therapy

Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets. *Usual dosage:* One or two 400 mg. tablets t.i.d.

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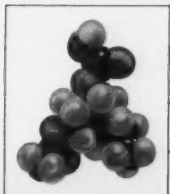
tranquilizer with muscle-relaxant action

2-methyl-2-n-propyl-1,3-propanediol dicarbamate



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*Belafsky, H. A., Breslow, S. and Shangold, J. E.: Meprobamate in pregnancy. Obst. & Gynec. 9:703, June 1957.

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STOP respiratory congestion orally

relief in minutes..lasts for hours

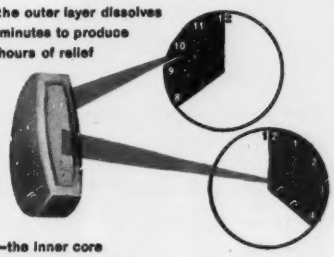
In the common cold, nasal allergies, sinusitis, and postnasal drip, one timed-release Triaminic tablet brings welcome relief of symptoms *in minutes*. Running noses stop, clogged noses open—and *stay* open for 6 to 8 hours. The patient can breathe again.

With *topical* decongestants, "unfortunately, the period of decongestion is often followed by a phase of secondary reaction during which the congestion may be equal to, if not greater than, the original condition. . . ."* The patient then must reapply the medication and the vicious cycle is repeated, resulting in local overtreatment, pathological changes in nasal mucosa, and frequently "nose drop addiction."

Triaminic does not cause secondary congestion, eliminates local overtreatment and consequent nasal pathology.

*Morrison, L. F.: Arch. Otolaryng. 59:48-53 (Jan.) 1954.

Each double-dose "timed-release" tablet keeps nasal passages clear for 6 to 8 hours—provides "around-the-clock" freedom from congestion on just three tablets a day


first—the outer layer dissolves within minutes to produce 3 to 4 hours of relief
then—the inner core disintegrates to give 3 to 4 more hours of relief

Each double-dose "timed-release" TRIAMINIC *Tablet contains:*

Phenylpropanolamine hydrochloride	50 mg.
Pyrilamine maleate	25 mg.
Pheniramine maleate	25 mg.

Dosage: 1 tablet in the morning, afternoon, and in the evening if needed.

Also available: Triaminic Syrup, for children and those adults who prefer a liquid medication.

Triaminic®

"timed-release" tablets

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The exfoliative cytological examination is called by some doctors the *cytologic cervical test*—by others the *"Pap" smear test.* In urging all women to have this test annually, we are calling it the *cell examination for uterine cancer.*

Here are our reasons:

Cytologic cervical test is a term which seems complicated to many women.

"Pap" smear test is simple, but women we have talked to find the word "smear" unpleasant and disturbing, and it may add to their anxieties about pelvic examinations.

Public relations advisors say that broadcasters and editors will dislike "smear"– and TV, radio and the press will be essential to the success of this educational project.

We have considered other terms but have at last agreed on *cell examination for uterine cancer* as the term which simply and accurately describes the keystone of this vitally important program.

This test can help save thousands of women each year. In many parts of the country it is becoming widely accepted as a part of a routine checkup. As fast as county medical societies approve, our local Units will urge women to go to their physicians annually for a *cell examination for uterine cancer.*

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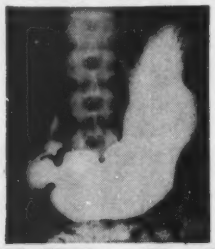

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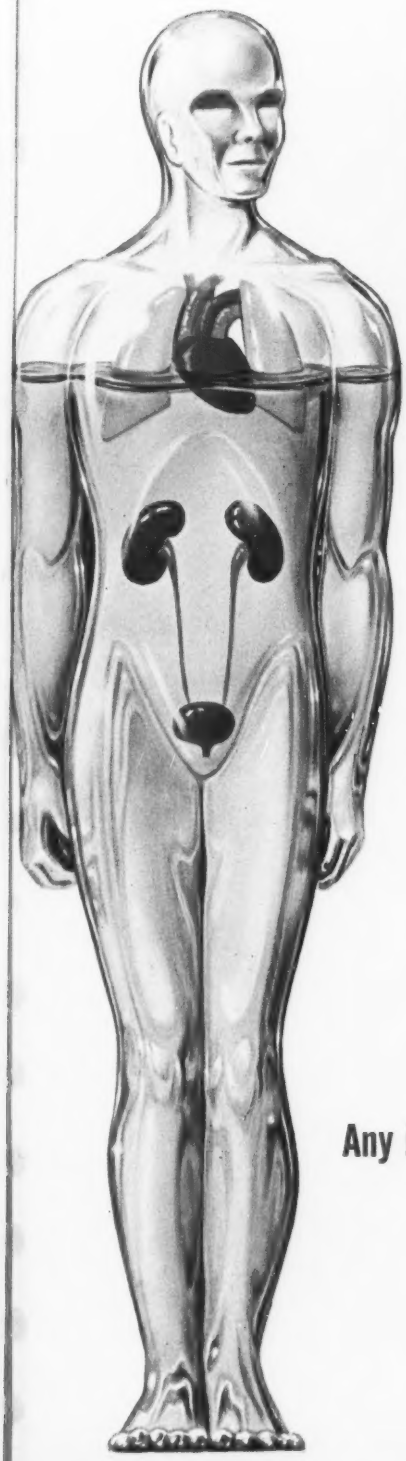

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CHLOROTHIAZIDE

an orally effective nonmercurial agent with diuretic activity equivalent to that of the parenteral mercurials

1 Gm. of 'DIURIL' orally is approximately equivalent to 1 cc. of mercurial I.M.[1]

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balanced excretion of sodium and chloride

Even in the presence of severe renal, cardiac or hepatic damage—

Any indication for diuresis is an indication for 'DIURIL':

1. Congestive heart failure of all degrees of severity
2. Premenstrual syndrome (edema)
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4. Renal edema—nephrosis; nephritis
5. Cirrhosis with ascites
6. Drug-induced edema

   May be of value to relieve fluid retention complicating obesity

MSD
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DIURIL is a trade-mark of Merck & Co., INC.

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enhances markedly the effects of the antihypertensive agents

reduces dosages of other agents below the level of serious side effects

smoothes out blood pressure fluctuations[1,2]

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Supplied: 250 mg. and 500 mg. scored tablets 'DIURIL' (chlorothiazide), bottles of 100 and 1000.

References

1. Hollander, William, and Wilkins, Robert W.: Chlorothiazide: A New Type of Drug for the Treatment of Arterial Hypertension, Boston Medical Quarterly *8*:69 (Sept.) 1957.

2. Freis, Edward D., and Wilson, Ilse M.: Potentiating Effect of Chlorothiazide (DIURIL) in Combination with Antihypertensive Agents, a Preliminary Report; Med. Annals of the District of Columbia *26*:468 (Sept.) 1957.

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a major breakthrough in the management of two major medical problems

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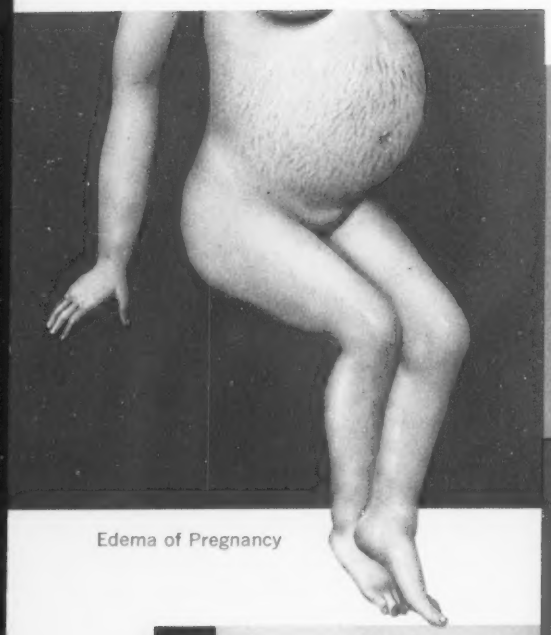

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REFERENCES:

1. Moyer, J.H., Ford, R.V., and Spurr, C.L.: Pharmacodynamics of Chlorothiazide (Diuril), An Orally Effective Non-Mercurial Diuretic Agent, Proc. Soc. Exper. Biol. and Med. *95*:529 (July) 1957.

2. Ford, Ralph V., Handley, Carroll, Moyer, John H., and Spurr, Charles L.: Chlorothiazide, An Orally Effective Non-Mercurial Diuretic Agent, Med. Rec. and Ann. *51*:376 (April) 1957.

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in spastic and irritable colon

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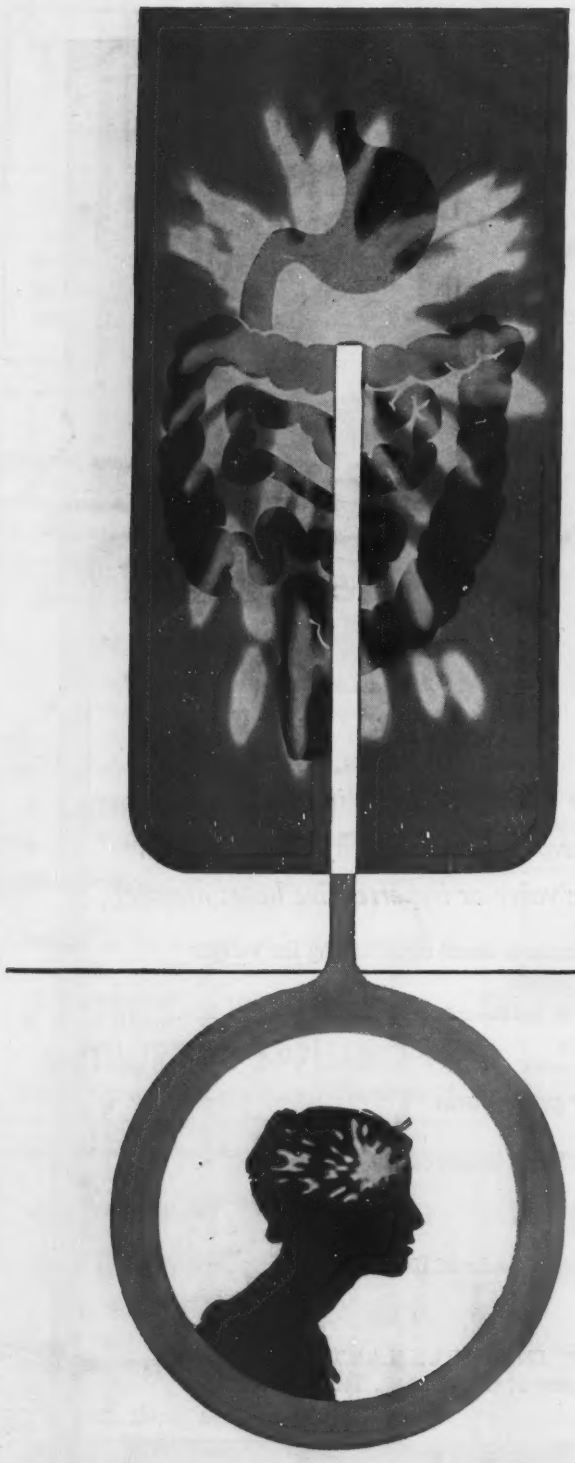

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